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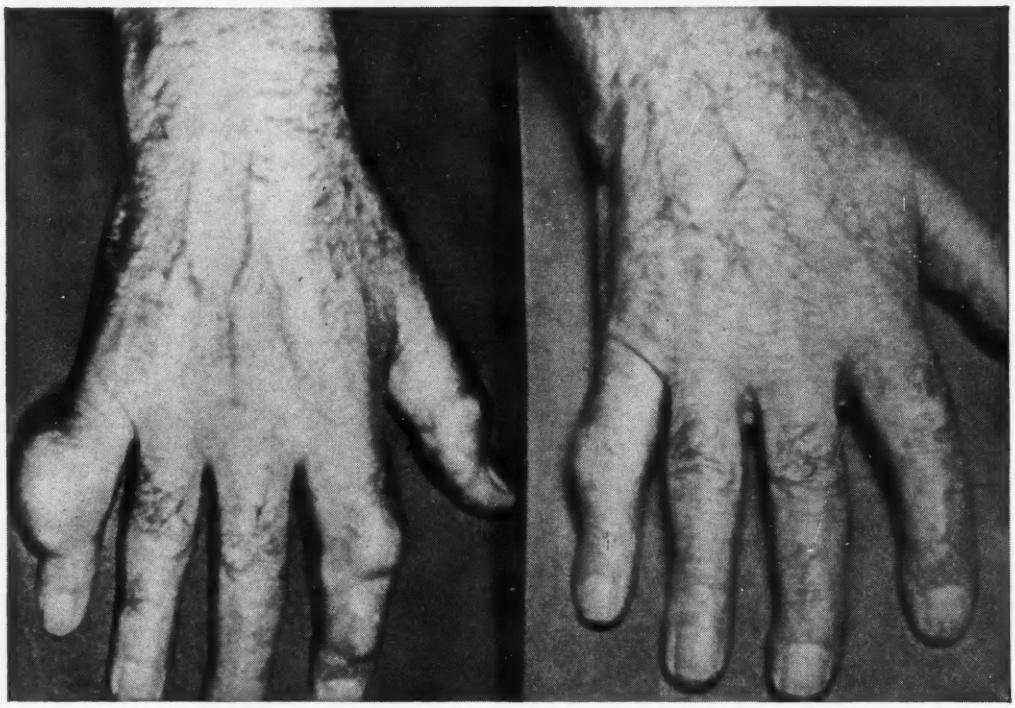

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The Status of Corticosteroid Therapy In Dermatology

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• *Therapy with systemic corticosteroids, despite attendant serious risks, is mandatory in diseases such as pemphigus, acute disseminated lupus erythematosus and some cases of exfoliative dermatitis that are ordinarily fatal, for in such cases life may be prolonged and the patients made comfortable. If no contraindications exist, therapy with corticosteroids is desirable, for diseases of short duration—contact dermatitis, serum sickness reactions and drug eruptions of all kinds—provided the causative factors have been removed and the reactions are causing severe distress.*

On the basis of encouraging reports in the literature corticosteroid therapy may be instituted with justification for a group of unrelated, intractable and discomforting diseases such as maddening pruritus ani, sclerema neonatorum, dermatomyositis, certain cases of sarcoidosis, berylliosis, Behcet's syndrome, universal calcinosis, Reiter's disease and ulcers of sickle-cell anemia.

One must always bear in mind the well-defined contraindications to corticosteroid therapy and the hazards of its use, particularly if therapy is to be prolonged.

Results from topical hydrocortisone therapy are particularly pleasing in chronic eczematous otitis externa and especially when it is combined with an antibiotic drug. Results are excellent also in nuchal eczema, dermatitis of the eyelids and in pruritus ani.

More often than not, hydrocortisone ointment and lotions benefit more than do other standard remedies such diseases as atopic eczema, contact dermatitis, lichen simplex-chronicus and eczematized phases of conditions such as psoriasis and superficial mycotic infections. Preparations containing a combination of hydrocortisone and an antibiotic are more useful than hydrocortisone alone.

When used with discrimination, with full attention to the selection of cases and proper concentration in the correct vehicle, hydrocortisone preparations in combination with antibiotics are excellent antieczematous agents.

IT IS NOW approximately five years since the corticosteroids were first made available for general use and specifically recommended for the treatment of rheumatoid arthritis. Very soon, and understandably, steroid therapy was extended to the so-called relatives of arthritis—the collagen group of diseases and diseases of hypersensitivity. And so it was that dermatology in particular felt the impact of this new wonder therapy. As steroid therapy was rapidly extended to more and more diseases, it soon became evident that the therapy had not only assets but limitations and liabilities. Because the drugs are capable of producing profound systemic reactions—some tragic in their consequences—there is still considerable disagreement as to the justification for

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their use for certain conditions. Yet in general they are being employed with ever increasing frequency and for more and more diseases, so that, as some one waggishly remarked recently, "Physicians may be divided into three classes: Those who use ACTH or cortisone for everything, those who never use them for anything, and those who use them only when they are needed."

It would seem that sufficient time has passed to permit the formulation of definitive indications. However, this has proved difficult because of the fact that, although remarkable progress has been made in the fundamental knowledge of the steroids, so much still remains unknown of their actual mode of action. There is still no full explanation as to why one drug is sometimes more effective than the other; still no way of formulating precise dosage; and still insufficient knowledge of the full effects or hazards from prolonged therapy. It would seem that a workable plan could be formulated, for in general the corticosteroids have proved a boon for skin diseases of three general groups: Diseases such as pemphigus or acute disseminated lupus erythematosus that are usually fatal; diseases such as crippling eczema that are not fatal but which may ruin life; and diseases such as discomforting drug eruptions or widespread acute poison ivy dermatitis which are of short duration provided the causative factors have been removed.

There is general agreement that corticosteroid therapy, when combined with both supportive measures and with antibiotic agents to control infection, constitutes the best therapy yet devised for the treatment of pemphigus. Patients with pemphigus are alive today who undoubtedly would not be had corticosteroids not been available. Large dosages are often necessary for patients with pemphigus—as large as 1,000 mg. or more a day of cortisone for a while—and, of course, therapy has to be maintained for long periods despite the risks of Cushing's syndrome, diabetes, psychosis and fractures. These constitute calculated risks which seem important when life itself is at stake, and beneficial effects from the drugs in such cases have thus far greatly outnumbered the incidence of distressing effects. Usually patients with pemphigus respond alike to corticotropin, cortisone or hydrocortisone; but in some instances, and for some unexplained reason, one drug proves effective where the others fail.

Perhaps the most dramatic results from this therapy occur in patients with acute systemic lupus erythematosus, but not in the chronic or subacute forms of the disease. There have been innumerable reports of moribund patients coming to life after a day or two of therapy. The author's first experience with such a case left an indelible impression. The patient was a youngster in her teens who was sadly close to death. The hospital had just received a new drug called ACTH—one of the first supplies of the drug to be released five years ago for experimental purposes only. It was administered to the girl, who was hopelessly moribund, and within 48 hours she was sitting up in bed, talking and alert. At last report, not long ago, she was still in fairly good health. After an experience like that it would be difficult to withhold such treatment from patients with acute systemic lupus erythematosus even though the drugs effect no cure but merely suppress symptoms—and, as with pemphigus, patients eventually die of the disease.

The use of corticosteroid therapy, it would seem, is not only justifiable but often desirable in diseases with reactions of hypersensitivity that are usually self-limited, such as severe drug eruptions, acute angioneurotic edema, erythema multiforme or dermatitis venenata. If the causative factor has been removed (drug discontinued, irritating plant or chemical discarded), quick relief from discomfort can usually be attained by corticosteroid therapy in sufficient dosages. If the eruption is severe, cortisone (or comparable amounts of corticotropin or hydrocortisone) given on a schedule of 300 mg. in divided dosages for 24 hours followed by 200 mg. for a day or two, and then decreasing by 25 or 50 mg. each day, usually effects gratifying relief to the patient. And the entire treatment is complete within a week or two so that risks from it are lessened considerably.

It is with the third group of diseases that a good deal of soul-searching is required before instituting corticosteroid therapy—that group of chronic, nonfatal diseases such as atopic eczema or eczematized psoriasis that become so acute or widespread as to constitute a life-ruining or crippling disease. Long-term therapy is usually necessary for such diseases and the hazards of prolonged therapy may be such as to raise a question as to whether the treatment is justified. Certainly it would seem justified if in the physician's opinion the patient is actually incapacitated, for in such cases therapy for a short period would effect relief enough to restore the patient's usefulness, albeit for a relatively short time. But what of the patient who, because of the comfort obtained, understandably pleads for continued treatment although he can scarcely be classified as being incapacitated? Even the more intelligent patients in those circumstances tend to throw caution to the winds and it is then that the physician's position becomes difficult. But no matter how difficult, it is essential that he assume control of the situation just as he does in prescribing narcotic drugs for alleviating pain. Fortunately, investigators have studied the problem and have supplied a workable slide-

rule. In the field of dermatology, Sulzberger and co-workers[4] recently reported their observations on a group of 35 patients with chronic and recurrent skin diseases in which they attempted to ascertain whether or not prolonged therapy affected three things: either increased tolerance or diminished therapeutic effectiveness; addiction; or ill effects higher in incidence or different in kind from short term medication. It would seem from their experience that long-term therapy for chronic recurrent eczematous eruptions may be instituted justifiably as a calculated risk provided certain precautions are observed—of which more later. Their procedure was to institute dosages large enough—cortisone up to 300 mg. a day in divided doses—to allay symptoms quickly. If necessary the dosage was increased unhesitatingly but then, in order to avoid a rebound-reaction, they reduced it by "feeling" their way, for there is not yet a mathematical guide to dosage. If possible the dosage should be brought below 100 mg. daily relatively soon, for the incidence of reactions tends to increase when doses are higher than 100 or 125 mg. daily. As was to be expected, frequently it was found necessary to increase the dosages for a while in order to care for flareups, but it was found, too, that many of the patients were kept comfortable by dosages as low as 5 mg. of cortisone every second day.

Should the experience of additional observers parallel those of Sulzberger and co-workers—and it appears that they will—it is quite likely that long-term therapy for discomforting diseases may become more widely used despite the misgivings of some experienced investigators. Such misgivings are well founded, for corticosteroid therapy is capable of masking hidden active infections and it may reduce fever and maintain a feeling of well-being in the face of serious infections and destruction of tissue. Some of these unfortunate complications may not manifest themselves until some time after the steroid therapy is begun. It may render perforation of a viscus painless. Such therapy may also produce convulsions and psychotic reactions, severe headache, edema, glycosuria and the oft-mentioned Cushing's changes of moon facies, hirsutism, adiposity, striae, acne and buffalo neck. Fortunately these changes are usually reversible but not the disastrous fractures from the relatively rare osteoporosis.

For such reasons it is essential never to institute corticosteroid therapy unless first ascertaining whether or not the patient has or has had diseases such as diabetes, hypertension, peptic ulcer, pulmonary tuberculosis or personality disorders. If the therapy is to be used for long, it is well to determine the blood cell count, the serum sodium, potassium and calcium levels and the fasting blood sugar level, and to have roentgenograms of the long bones for evidence of decalcification. The patient should have a low salt diet (potassium chloride 4 gm. daily) and weekly blood pressure determinations, weight measurements and urinalysis.

It was inevitable, of course, that a therapy capable of producing dramatic results would be used for many and varied diseases, particularly the hitherto intractable diseases. Corticoids have been tried and found useful for the treatment of herpes gestationes but not for herpes simplex or herpes zoster. In fact, herpes zoster occasionally occurs in patients undergoing treatment with steroids for other conditions. Acute lichen planus, but not the chronic form, is helped by corticosteroids, as is alopecia totalis, but this only for as long as the therapy is being used. For the latter condition one must weigh carefully the possible benefits from therapy against the risks, for therapy might conceivably be a life-long matter. Recalcitrant eruptions of the palms and soles is helped sometimes but not always; and other conditions said to be helped on occasion—but not in all cases—are scleroderma, dermatomyositis, dermatitis herpetiformis, sarcoidosis, intractable pruritus ani, sclerema neonatorum, keratosis blenorrhagicum, berylliosis, Behcet's syndrome, universal calcinosis, incontinentia pigmenti, Reiter's disease and ulcers of sickle cell anemia. Corticosteroid therapy has been highly recommended for the control of generalized pruritus of Hodgkin's disease and it is used also as adjunctive treatment to other therapies for lymphoblastomatous disease. Undoubtedly if all reports were in, the list of diseases treated by corticosteroids would be much longer.

TOPICAL THERAPY

When used topically for the treatment of skin diseases, cortisone was found to be ineffective, but hydrocortisone, which was introduced in 1952, has definite value in the treatment of certain of them. At first the short supply and high cost of the drug limited study to relatively few centers, but the first reports were so encouraging that there are now, according to Witten,[5] more than 35 different preparations available, composed of six chemical forms of hydrocortisone. They are incorporated in some 14 different bases, put up in various concentrations from 0.1 per cent to 5 per cent including six preparations in combination form with antibiotics. Available on the market are preparations of hydrocortisone acetate, free alcohol hydrocortisone and fluorhydrocortisone acetate. Other preparations such as chlorhydrocortisone acetate and hydrocortisone cyclopentyl propionate and fluorhydrocortisone free alcohol are being investigated.

The results from treatment with topical hydrocortisone acetate parallel closely those obtained from

systemic treatment with corticosteroids. They act to block tissue reactions to disease-producing factors. Hence their greatest value is to reduce inflammatory eczematous reactions of various kinds, such as atopic eczema, eczema ani, contact dermatitis—particularly on the eyelids—lichen simplex chronicus and sometimes seborrheic eczema. As occurs with systemic therapy, chronic diseases in general soon relapse when the ointment is discontinued and on occasion rebound so actively as to leave the patient in a condition worse than before therapy. On occasion, too, a most satisfactory result from its use is observed, particularly in atopic eczema where the symptoms are suppressed as long as the ointment is used. Then there are instances in which beneficial results would be expected from the therapy but the results are disappointing.

It was feared at first that the topical application of hydrocortisone, like systemically used corticosteroids, would be dangerous in the presence of secondary bacterial or mycotic infections. This has not been borne out. In fact, ointments combining hydrocortisone and antibiotics have given superior results in superficial infections with the possible exception of herpes simplex. Primary irritation or sensitization-reactions from hydrocortisone ointment appear to pose no problem. In the few cases in which irritation has occurred it apparently was due to the base, but of most importance there has been to date no evidence of undesired systemic complications from relatively prolonged use of hydrocortisone acetate or free alcohol-type ointment in the dosages employed. As Lorincz[2] pointed out, however, because of its convenience and rapid symptomatic effects, many patients may be committed to hydrocortisone ointment who will become dependent on it for relief and who may continue to use it for long periods. The ointment has had extensive use for only a little more than a year so that the hazards to the skin from long-term use, if any, have not yet been determined in a sufficient number of cases. "As sign-posts of caution we should keep in mind the well known degenerative effects produced in connective tissue by intradermal hydrocortisone injections,[1] and the observations of Piccagli et al.[3] on the synergistic effect of systemic cortisone administration in mice on epidermal carcinogenesis induced with methylcholanthrene. Thus we might ask—what, if any, is the risk of inducing connective tissue degeneration and consequent premature aging of the skin by prolonged topical hydrocortisone therapy—and what, if any, is the risk of enhancing the development of malignant changes in the skin by such long-term use of topical hydrocortisone."

These risks, even if they are more theoretic than real, should have the attention of investigators, for the employment of hydrocortisone preparations for topical therapy is increasing constantly. Indeed, we are in a period of too-reckless use that may well result in disappointment with a drug that does have undoubted value if used with discrimination. To use it indiscriminately in place of other well established modalities must lead to disappointment. Such factors as the use of an ointment when a wet dressing is indicated, the failure to remove causative irritants when possible, neglect in employing a proper vehicle, failure to adjust to a proper dosage, all contribute to disappointing results. The use of hydrocortisone ointment on noneczematous lesions also constitutes indiscriminate use, for hydrocortisone ointment has no value when applied to common conditions such as psoriasis, discoid lupus erythematosus, lichen planus, alopecia areata, keratoses or warts.

We at Northwestern University recently completed a study of some 1,200 patients treated topically with hydrocortisone acetate in different concentrations and in various vehicles.* Whenever possible, simultaneously paired comparisons were made, using hydrocortisone preparation on one side of the body and the base—or sometimes an old standard-type ointment—on a similar lesion on the opposite side. The results were rather similar to those already noted in other reports. A good response from the ointment was obtained in approximately two-thirds of the cases of atopic and infantile eczema, in contact dermatitis and in localized neurodermatitis—a ratio just a little better than that obtained by older effective therapy. The effects were superior to older methods in dermatitis involving the eyelids, in nuchal eczema and especially in chronic eczematous otitis externa. In acute poison ivy dermatitis, hydrocortisone-antibiotic suspension effected excellent results, but not the ointment. And the suspension was effective also in eczematous eruptions involving the axillae and crural areas. The response to hydrocortisone ointment was highly satisfactory in anogenital pruritus, but more so in pruritus ani than pruritus vulvae. Hydrocortisone was less effective in nummular eczema, seborrheic dermatitis and infectious eczematoid dermatitis, although on occasions dramatic results were observed in all three of these conditions. It should be mentioned that favorable results were not invariably obtained in any disease, not even in contact dermatitis of the eyelids.

It was noted that a concentration of 1 per cent hydrocortisone acetate ointment in a base of liquid paraffin, petrolatum, cholesterol and multiwax was adequate for routine use. It was superior to 0.5 per cent ointment but occasionally was inferior to 2.5 per cent concentration. Sometimes a cream-type

*The materials for this study were supplied by the Upjohn Co., Kalamazoo, Mich.

base was more desirable than the ointment and the suspension was better for use on widespread areas, in hairy areas and in folds such as the axillae and groins. Preparations containing hydrocortisone and neomycin appeared to be a little more effective than hydrocortisone alone. No difference in the results as between the acetate and the free alcohol preparations was observed. Neither proved irritating. Both had only a localized action. The fluorohydrocortisone ointment about which there is at present some uncertainty as to whether it is free of systemic effect, was not used.

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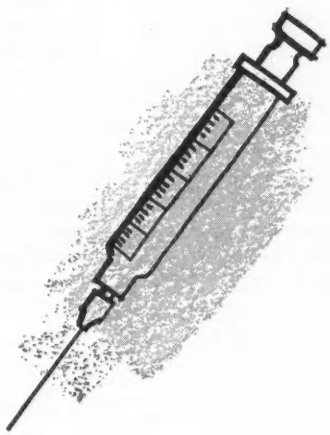

Acute Infections of the Gastrointestinal Tract

JOHN M. RUMSEY, M.D., San Diego

• *Even though "shot-gun" treatment often proves effective in acute enteric infections, attempt to establish accurate diagnosis is still worthwhile. In most cases acute enteric infection is self limited and responds well to symptomatic treatment. Postantibiotic diarrhea is relatively common and is often severe. Usually the organism is staphylococcus and at present, at least, erythromycin seems to be the drug of choice in treatment.*

Antibiotics should be used only when definitely indicated. Indiscriminate "specific" treatment for acute diarrheas may mask acute infections with organisms which are suppressed—not conquered. These infections present a potential public health problem.

Cases of acute gastroenteritis that are apparently of staphylococcic or viral origin should be managed symptomatically at first. If satisfactory response is not obtained quickly, accurate bacteriological diagnosis should be sought.

DURING THE LIFETIME of the present generation of American physicians, the concept of the relative importance of acute gastrointestinal infections has undergone great change. Some years ago, these infections represented a major public health problem and a vital phase of general medical practice in the country. In the past 20 years, however, enteric infections have assumed a position of less importance—first, because of the efforts of public health departments everywhere; second, because of widespread instruction in sanitation and pursuance of sanitary measures on both an individual and a community level; third, the use of anti-infectious agents; and fourth, the greatly increased availability of good medical care.

The favorable position of present-day physicians with regard to gastrointestinal infections is, nevertheless, a kind of double-edged sword. The patient, as well as the physician, is likely to treat a potentially serious infection much too casually. The majority of acute gastrointestinal problems consist of acute staphylococcus infection or toxemia—so-called "food poisoning." When a physician considers the differential diagnosis in these conditions, there are several possibilities, most of which look quite similar at the outset. There is usually a fairly acute onset of nausea and malaise, abdominal cramping, diarrhea, and frequently, vomiting. Rarely can specific stool characteristics permitting exact diagnosis be found. Differential diagnosis of acute diarrhea must, therefore, be established. Following is a list of 11 of the more common causes:

1. Acute staphylococcal infection.
2. Acute staphylococcal toxemia.
3. Postantibiotic diarrhea.
4. Acute shigellosis.
5. Acute salmonellosis.
6. Acute viral gastroenteritis.
7. Acute "touristas."
8. Amebiasis.
9. Acute nonspecific ulcerative colitis.
10. Acute exacerbation of irritable colon.
11. Acute diverticulitis.

Certainly the commonest is acute food poisoning—generally a staphylococcal gastroenteritis resulting from toxic products of staphylococcic growth. Clinically this disease is characterized by severe nausea and vomiting, with diarrhea and abdominal cramps. There may be moderate fever, malaise, occasionally severe prostration, and sometimes weakness and fainting. The patient may be quite ill at first, but the disease is, in general, self limited. As soon as the toxic products of bacterial growth are discharged and the irritability of the gastrointestinal tract is relieved, the patient recovers.

A substantial proportion of the cases of acute gastrointestinal disease seen by the average physician probably are cases characterized by the syndrome described. Each physician has his own method of managing it, and each probably believes his particular regimen is best. However it is done, symptomatic treatment usually meets with success. As a student, the author was told that the most rapid means of ridding the patient of toxic products was by saline purge, but the results by this method, on the few occasions it was tried, were singularly unimpressive as compared with results obtained by usual procedures. Many drugs suggested for use in the management of such cases involve, primarily, medication to alter gastrointestinal tract motility, adsorbents, ion-exchange resins,[6] opium, and other sedatives. When a patient has acute toxic vomiting, the author knows of nothing that will more quickly alleviate symptoms than a large dose of sodium phenobarbital with atropine, given intravenously and followed by kaolin

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preparation mixed with paregoric. The patient's diet should be limited for several days thereafter. Heat may be applied to the abdomen, and if fluid loss has been great, fluid and electrolyte replacement by parenteral feeding may be necessary. In some cases of staphylococcic gastroenteritis, an antibiotic—erythromycin—is required. It has proved most effective.

Staphylococcic gastroenteritis sometimes takes an acute fulminant form. In 1953 Terplan[15] reported an epidemic of acute fulminant staphylococcic gastroenteritis occurring postoperatively. Eight patients were affected and all died. The onset of this syndrome began with gastrointestinal upset, diarrhea, fever, shock, and a toxic state so severe and debilitating that the patient had little chance to react from it. The syndrome took place, in each instance, after operations differing in type each from the others, and death followed in three to eight days. All patients received antibiotics, apparently in rather small amounts—penicillin, streptomycin, aureomycin, terramycin and chloramphenicol. Conditions noted at autopsy were: Loosely adherent membrane, with some invasion of the mucous membranes, badly inflamed mucosa, and an obviously overwhelming growth of staphylococci. The staphylococci were of a coagulase-positive hemolytic strain. Since Terplan's report, a number of other similar cases have been identified. Apparently, heroic supportive measures are the only means of checking this disease, together with some agent to stop growth of the staphylococcus. (Dr. Terplan told the author that chloramphenicol is the only drug to which staphylococci were found never to be resistant. He said that, in England, many strains of coagulase-positive staphylococci were found to become resistant to erythromycin. The belief in this country is that erythromycin will conquer this type of infection.)

Kleckner, Bargen and Baggenstoss[10] reported cases similar to those reported by Terplan, that were diagnosed as severe acute pseudomembranous enterocolitis, and in which rapid progressive circulatory collapse occurred. These cases were quite resistant to supportive or resuscitative treatment, and irreversible shock and death took place in a matter of hours. The disease resembled Asiatic cholera, was associated with some fever, severe acute abdominal distention, congestive failure, vomiting and not much diarrhea. Dr. Terplan said he felt that this was not the same disease as that in his series.

As a rule the usual postantibiotic diarrhea[2,4,9,11] is owing to staphylococcic gastroenteritis, which may be considerably debilitating. The condition is characterized by watery diarrhea, abdominal cramping, loss of weight and often fever. Gastrointestinal disturbances, especially diarrhea, should be warning signals. If symptomatic treatment does not give instant relief, the antibiotic in use should be discontinued. If diarrhea should become intractable and staphylococcus aureus should grow on culture, erythromycin seems to be the present drug of choice. If staphylococcus aureus is suspected, the laboratory should be informed, so that suitable media may be used in the culture. Since postantibiotic diarrhea is a frequent and difficult problem, it seems clear that antibiotics should be used only when specifically indicated. In dealing with postantibiotic diarrheas, good results have been obtained with the use of lactic acid cultures and even buttermilk enemas.

Acute shigellosis,[4,5] although less frequent than before, is still an important cause of gastrointestinal infection. Now that potent antibiotics and chemotherapeutic agents are available, in many of the acute diarrheal diseases cultures are not made. Therefore, any present-day statistics on incidence of acute shigellosis are bound to be inadequate. Nevertheless, public health departments in every large city[3,12] report a substantial number of cases of this disease each year. It occurs in all age groups, but is more often reported in children. Although generally there is acute fever with diarrhea, shigellosis may be afebrile at the onset. One-third of a large group of children reported upon in a series from Chicago did not have fever in the early stage of the disease. If fever is present, it may last for more than a week. Stools may vary in type—some are bloody and some are not. The patients may have symptoms in areas of the body other than the intestinal tract—predominantly respiratory or central nervous system symptoms. When vomiting occurs, shigellosis is often impossible to differentiate clinically from food poisoning. Strangely, it affects food handlers frequently, creating a serious public health menace. The disease is diagnosed by culture.[8] Cultures of material removed by sigmoidoscope are most dependable, and cultures of the passed stool least so. Culture of a rectal swab seems the most practical method. Shigellosis is treated effectively[7] by streptomycin, both orally and parenterally, aureomycin, terramycin or sulfasuxidine.

Any discussion of acute gastrointestinal infections is not complete without mention of salmonellosis. Typhoid fever still strikes. Even though only a few cases crop up in the large cities each year. Physicians must be aware of it and attempt to diagnose it as early as possible. Although chloramphenicol seems the treatment of choice and an effective agent, typhoid fever remains a serious problem. The mortality rate is high. A recent report[1] from Mexico City described excellent results in the treatment of typhoid fever with use of synnematin B. In the preliminary study, synnematin B seemed to be a distinct improvement over chloramphenicol.

Other salmonella infections happen in all age groups.[3,12] They, like shigellosis, occasionally are afebrile in the beginning; fever for more than a week

is not unusual. Excessive diarrhea occurs more often than not, and there is evidence of respiratory and central nervous system symptoms. Salmonella, again similar to shigellosis, may be difficult to differentiate from acute food poisoning. This infection also affects food handlers often enough to create a public health hazard. The drugs of choice are chloramphenicol and sulfasuxidine.[7]

A somewhat nebulous but well enough defined clinical entity is viral enteritis.[4,14] In everyday practice, more cases of acute gastroenteritis are attributed to viral infection than to any other cause. Viral enteritis ordinarily is epidemic and mild, associated with nausea, diarrhea, abdominal cramping, low grade fever, malaise and coincident coryza. The disease limits itself as a rule, and the treatment of choice is again symptomatic. How often viral enteritis takes place is hard to determine. Certainly well-documented epidemics of it have been reported. When it is manifest in sporadic bouts, it is difficult to diagnose and difficult to differentiate from a nonspecific irritable colon disturbance secondary to respiratory infection. It is well known that respiratory infections commonly excite flare-ups of ulcerative colitis and peptic ulcer, as well as irritable colon. There might be some direct connection between these episodes and disease directly involving the gastrointestinal tract.

"Montezuma's revenge," the acute gastroenteritis experienced by many travelers in Mexico, affects travelers in many countries besides Mexico. Interestingly, a Mexican physician has told the author that Mexicans frequently have similar upsets while traveling in the United States, or from one part of Mexico to another. The travelers' acute gastroenteritis is a clearly defined entity that occurs so often that most travelers would rather embark without a camera than without a bottle of paregoric. The cause of this syndrome has been the subject of much discussion and heated argument from one country to another. Many things have been indicted as etiologic possibilities, but it is likely that the cause is a combination of factors. Olive oil, foods cooked in olive oil, sesame oil used in cooking, herbs, peppers and tomatoes have all been mentioned as potential troublemakers. Staphylococcus contamination makes a major contribution. Many tourists have previously assaulted their gastrointestinal tracts with large quantities of alcohol, and sometimes the alcohol itself may account for the acute problem. If not, it adds to the difficulty. In the same vein, fatigue connected with travel and entertainment incident to travel can contribute to preparation of the individual for the gastrointestinal cataclysm. Allergic sensitivity to food is a possibility, since people on vacation will throw caution to the winds and attempt things they would not think of at home. The author believes that amebiasis seldom produces the "tourista" syndrome. The course is self limited and sequelae practically nonexistent—which would not be the case if so many people with amebiasis were treated symptomatically.

The clinical features of travelers' gastroenteritis are acute nausea, acute abdominal cramping, moderately explosive diarrhea (seldom, if ever, bloody), fatigue and mild prostration. The disease usually, but not always, limits itself. Treatment once again is symptomatic. Paregoric mixtures are the most essential drugs, although parenteral sedative, such as sodium phenobarbital, may be required when severe and protracted vomiting takes place. Dietary limitation to the blandest of foods for a short period of time is mandatory, as is a period of rest. The rationale for using drugs of the hydroxyquinoline group for instance, vioform—escapes me, although it does have a number of proponents.

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Electrolyte Balance in Gastrointestinal Disease

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• *Even small losses of gastrointestinal secretions when combined with reduced intake of electrolytes may seriously disturb electrolyte balance. Knowledge of the ionic composition of secretions lost is essential in planning therapy. Loss of gastric contents usually results in excessive loss of chloride; in achlorhydria this is not the case. Loss of sodium and potassium may be large in either case and is often underestimated. Small bowel obstruction results in a more balanced loss of electrolyte which may not affect acid-base balance greatly. In diarrhea loss of base predominates, and may result in a large potassium deficit. Steatorrhea due to nontropical sprue results in large fecal losses of sodium, potassium and chloride, in addition to the large calcium and phosphorus loss. In chronic peptic ulcer excessive ingestion of milk and absorbable alkalies may result in hypercalcemia, azotemia and alkalosis, without hypercalciuria.*

Since renal function is usually adequate in the milder gastrointestinal disturbances, electrolyte and fluid replacement should be started early, and can be guided by generally available laboratory tests, the carbon dioxide combining power and serum chloride levels, provided the predominate ionic loss is known and potassium deficiency remedied. If this is done, development of serious fluid and electrolyte deficits can usually be prevented.

THE LOSS OF LARGE VOLUMES of gastrointestinal secretions rapidly depletes body water and electrolytes. Serious complications rapidly result, and the need for prompt replacement therapy is evident. Milder disturbances of reabsorption in the gastrointestinal tract, however, result in loss of fluid and electrolytes which may not be promptly recognized. Although the degree of loss may not seem alarming, when combined with the reduced intake which almost always is concomitant significant depletion slowly but surely results. In such circumstances physicians are likely to defer expensive laboratory studies, expecting that the patient will improve, and indeed may aggravate the situation by either further diluting the body's electrolytes or administering excessive amounts of sodium chloride. However, if it is recognized that this problem is developing, and if the electrolyte content of secretions that are lost is known, correct replacement can be begun before signs and symptoms of disturbed electrolyte balance appear. In many cases this will prevent development of serious deficiencies of fluid and electrolytes and will hasten recovery.

In healthy persons total body water approximates 60 per cent of the body weight. With obesity the proportion drops, to as low as 42 per cent in the very obese.[5] Two-thirds of the body water is intracellular and one-third extracellular. Of the body weight approximately 40 per cent is intracellular water and 20 per cent extracellular, the latter being further divided into interstitial fluid and plasma, 15 per cent and 5 per cent respectively (see Chart 1).

Electrolytes in body fluids are now known to have two important functions. Inorganic ions play an important role in the specific function of cells such as nerve and muscle, and act as enzyme catalysts in metabolic processes. Cell membranes are no longer thought to be impermeable, but rather to be the site of continuous ionic interchange, with concentration gradients maintained by intracellular metabolic processes.[7] However, the older static concept of ions maintaining proper acid-base balance and controlling the distribution of body fluid is still useful in understanding the principles of replacement therapy.

Part of a Symposium on Disorders of the Intestinal Tract. Presented before the Section on General Practice at the 84th Annual Session of the California Medical Association, San Francisco, May 1-4, 1955.

Electrolytes in solution dissociate to form charged particles called ions. Positively charged ions, or cations, found in body fluids are sodium^{+}, potassium^{+}, calcium^{++} and magnesium^{++}. Negatively charged ions, or anions, are chloride^{-}, phosphate^{--}, bicarbonate^{-}, organic acids and proteins. Acid-base balance depends upon the number of acidic and basic ions present, since one basic ion will neutralize one acidic ion, even though the actual weights are different. To clearly express this activity, the unit milliequivalent is used. For a monovalent ion the equivalent weight is the atomic weight, but for a bivalent ion, which can neutralize two monovalent ions, the atomic weight equals two equivalents. Since weights of electrolytes are expressed in milligrams per 100 cc. of body fluid, the milliequivalent is used to permit use of whole numbers. To convert

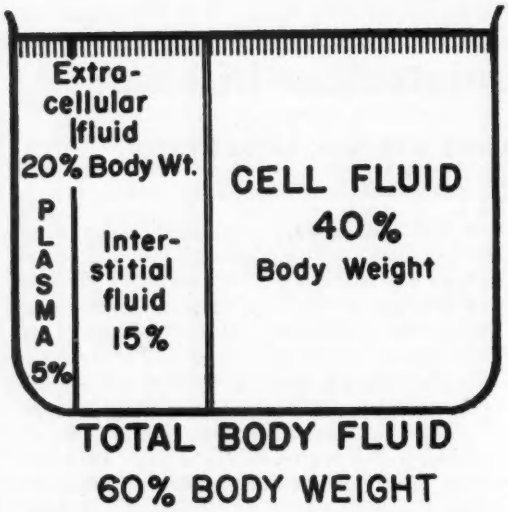

Chart 1.—Fluid compartments of the body (from Statland, H., Fluid and Electrolytes in practice, Lippincott, Philadelphia, 1954).

values in milligrams into milliequivalents the following formula is used:

MILLIEQUIVALENTS

$$\frac{\text{mg. \%}}{\text{atomic weight}} \times 10 \times \text{valence} = \text{mEq./L}$$

The chemical composition of extracellular and intracellular fluid is quite different in respect to sodium and potassium (Chart 2). The plasma protein content is the important difference between plasma and interstitial fluid. Interstitial fluid contains 138 mEq. per liter of sodium, 108 mEq. per liter of chloride, and 27 mEq. per liter of bicarbonate. In comparison, intracellular fluid contains 157 mEq. per liter of potassium, 26 mEq. per liter of magnesium, 110 mEq. per liter of phosphate and 74 mEq. per liter of protein. When serious electrolyte depletion occurs, the concentrations of sodium and potassium are altered, which interferes with cellular metabolism and in extreme cases may cause death.

Distribution of body water also depends upon the number of particles in solution, since water moves through a semipermeable membrane to the side containing the more concentrated solution until the solutions are equal in concentration. This shift is called osmosis. Osmotic force has no relation to chemical activity, or valence. Therefore, for an electrolyte the unit of osmotic pressure, or osmol, is that of a molar solution, or the gram atomic weight of the electrolyte per liter of solvent. To convert from milligrams per 100 cc. of body fluid to milliosmols (mOsm.) per liter, therefore:

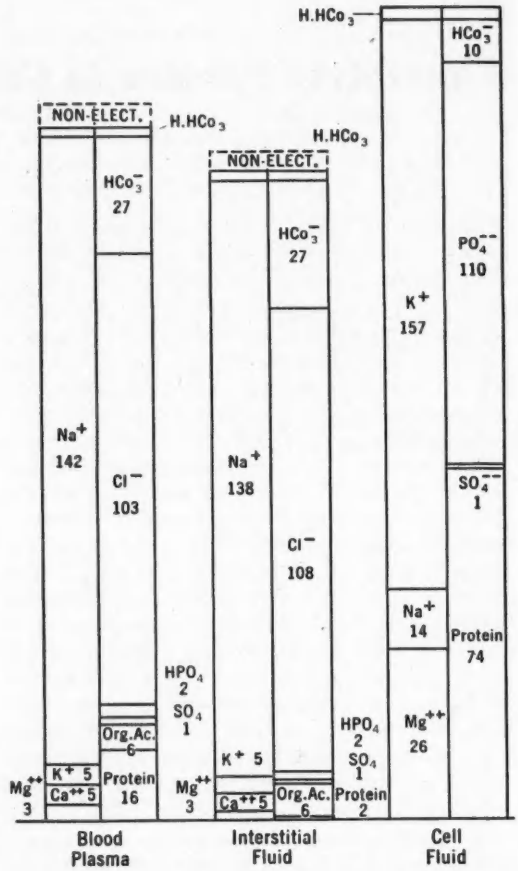

Chart 2.—Electrolyte composition of plasma, interstitial fluid and intracellular fluid (from Hardy, J. D., Fluid Therapy, Lea & Febiger, Philadelphia, 1954, and Bland, J. H., The Clinical Use of Fluid and Electrolyte, W. B. Saunders Co.).

MILLIOSMOLS

$$\frac{\text{mg. \%}}{\text{atomic weight}} \times 10 = \text{mOsm./L}$$

Since most of the ions present in body fluid are monovalent, the total of milliequivalents of anions and cations is a reasonable approximation of milliosmols present. Normally milliequivalent of chloride plus milliequivalents of carbon dioxide combining power equal 130 per liter. These determinations can be made in most laboratories, and are an important guide in therapy. If the total is less than 125 milliequivalents, osmolarity is decreased and more electrolyte is needed; if over 135, more water is needed to restore normal balance.[5]

An idea of the magnitude possible in abnormal loss from the gastrointestinal tract can be gotten by

considering the total 24-hour volume of gastrointestinal secretions:

Gastrointestinal Secretions (from Hardy,[5] p. 45)	Approximate Volumes ml./24 hours
Saliva	1,500 cc.
Gastric juice	2,500 cc.
(5 to 6 L. may be aspirated in pyloric obstruction)	
Bile (hepatic)	500 to 1,000 cc.
Pancreatic juice	900 cc.
Intestinal juice	3,000 cc.
	8,400

The total of 1,500 cc. of saliva, 2,500 cc. of gastric juice, 500 to 1,000 cc. of bile, 900 cc. of pancreatic juice, and 3,000 cc. of intestinal juice is almost two and a half times normal plasma volume. Any considerable loss results in rapid changes in the body's fluid and electrolyte distribution. Added to this is the daily obligatory fluid loss of 1,500 cc. —900 cc. through respiration and the skin, and a minimum urine volume of 600 cc. In correcting deficits the body's needs should first be calculated separately, that is, nutritional needs (usually glucose or fructose), electrolyte needs, and fluid needs, so the correct solutions to make up the total deficit may be administered.

Clinically loss of saliva and gastric juice through vomiting, pyloric obstruction or gastric intubation is frequently encountered. The loss of potassium and sodium is often underestimated. An important contributory factor is the failure of the body to maintain a positive potassium balance when intake is restricted,[2] showing that the body cannot conserve potassium as it does sodium. The electrolyte content of saliva and gastric juice reveals a mean sodium content of over 40 mEq. per liter for both, and higher values for potassium and calcium in saliva—20.4 and 6.5 mEq. per liter, respectively, as compared with 11.6 and 3.6 mEq. per liter in gastric juice.[1] Gastric juice potassium values significantly above normal were noted in persons who were hyposecretors or achlorhydric.

Studies on cases of pyloric obstruction due to peptic ulcer have shown a large chloride loss in excess of sodium[6]:

Pyloric Obstruction—mEq. per liter

	Peptic Ulcer Range	Peptic Ulcer Mean	Achlorhydric (Cancer) Range	Achlorhydric (Cancer) Mean
Chloride	76-157	126	39-110	58
Sodium	21-82	56	24-91	61
Potassium	6.5-26	12.6	7.1-24.6	10.2

On Levine tube drainage the mean chloride value was 126 mEq. per liter. In the achlorhydric patients with carcinomatous pyloric obstruction the chloride loss was still large, in the form of sodium and potassium chloride. The mean chloride value in these cases was 58 mEq. per liter. Mean values for sodium and potassium were about equal in the patients with

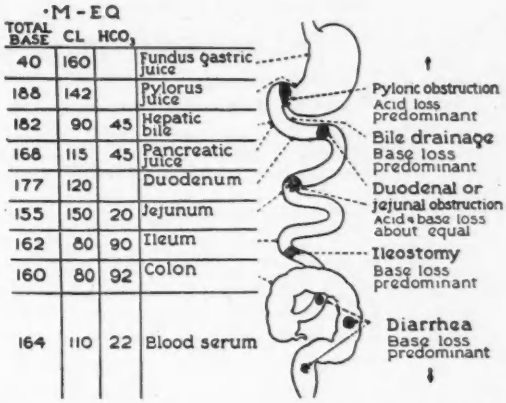

Chart 3.—Ionic composition of fluids at various levels of the gastrointestinal tract (from Coller, F. A., and Maddock, W. G.: Water and electrolyte balance, Surg., Gyn., & Obs., 70:340, Feb. 1940).

ulcer and in those with achlorhydria, being near 60 mEq. and 10 mEq. per liter respectively. Some patients with a loss of 3 or 4 liters a day lost 78 to 104 mEq. of potassium, equivalent to the potassium in 6 to 8 grams of potassium chloride. A hypochloremic hypopotassemic alkalosis results, which will not respond to treatment unless adequate amounts of potassium are given. If renal function is normal this can safely be done by adding 3 grams (40 mEq.) of potassium chloride per liter of fluid. Osmolarity may be relatively unaffected if loss of salts and water has been proportionate. Since electrolyte loss usually exceeds water loss,[5] the resulting hypotonicity may require hypertonic saline solution (3 per cent) for correction.

In cases of obstruction high in the small bowel, the increased sodium and potassium content in bile, pancreatic juice and intestinal juice balances the chloride excess usually found in gastric juice (Chart 3). Consequently severe reduction of the extracellular fluid may occur without pronounced disturbance of osmolarity or acid-base balance. In these situations isotonic electrolyte solutions are used for replacement, with particular attention to the potassium requirement.

Loss of upper small bowel or pancreatic juice through a fistula poses a difficult problem and cannot be handled properly unless accurate electrolyte determinations can be done. Usually a large volume of fluid with high concentrations of sodium, potassium and bicarbonate is lost.

In the lower intestinal tract secretions there is a higher concentration of base. Hardy[5] pointed out that in low small bowel obstruction the dilated bowel may hold 5 to 10 liters of fluid. In acute diarrhea loss of sodium and potassium may produce acidosis, and the loss of up to 100 mEq. per liter of potassium

TABLE 1.—*Milk-alkali syndrome*

1. Hypercalcemia without hypercalciuria or hyperphosphatemia.
2. Normal serum alkali phosphatase.
3. Renal insufficiency with azotemia.
4. Mild alkalosis.
5. May have calcinosis (eyes, subcutaneous).
6. Rapid improvement when milk and absorbable alkali withheld.

in diarrheal stools[9] often produces a large potassium deficit. Fortunately, renal function is usually normal in these cases, so if adequate urine volume is maintained it would seem safe to give from 80 to 120 mEq. of potassium daily for replacement and maintenance, thus preventing the development of a serious deficit.

In steatorrhea due to nontropical sprue, the increase in fecal calcium and phosphorus has been ascribed to the faulty absorption of fat in the small bowel. Finding an occasional case with acidosis and low serum levels of sodium and potassium during an acute exacerbation suggested that a broader defect of absorption existed. Comfort and co-workers[3] found that fecal losses of sodium, of potassium and of chloride might be as high as fifteen times, two times and five times normal values, respectively. Large fecal losses of calcium, phosphorus and nitrogen were also found. Serious electrolyte deficiencies may result. Corticosteroid therapy seems to favorably influence intestinal absorption in such cases, and present opinion is that this disease involves defective absorption of many things in addition to fat.

Pronounced disturbance of electrolyte balance is occasionally seen in cases of peptic ulcer when large quantities of milk and absorbable alkali have been ingested over a prolonged period of time. The disturbance is called the milk-alkali syndrome, and characteristic findings are: Elevated serum calcium level; calcinosis (most frequently present as an ocular lesion resembling band keratitis); renal insufficiency with azotemia; mild alkalosis; normal serum phosphorus and alkaline phosphatase, and lack of increased urinary excretion of calcium[4, 8] (see Table 1). When patients with this syndrome are put on a low calcium diet and given aluminum hydroxide in place of absorbable alkalies, prompt improvement of the azotemia and hypercalcemia results. This ordinarily distinguishes these cases from primary hyperparathyroidism, where hypercalcemia and hypercalciuria persist on a low calcium intake. In some cases, however, the differentiation may be extremely difficult.

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Functional Gastrointestinal Disturbances

Recognition and Treatment

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• *The recognition of functional gastrointestinal diseases depends essentially on certain positive features characteristic of them. When there are evidences of associated functional disturbances in other organ systems or in the patient as a whole, or characteristic clinical syndromes are present, and there is lack of symptomatic or objective evidence of organic disease on careful examination, the diagnosis of functional gastrointestinal disorder is likely.*

Treatment of functional gastrointestinal disturbances rests fundamentally on the art of medicine in the treatment of the patient and not on the science of medicine in the treatment of a disease. The essential steps in successful treatment include convincing the patient of the diagnosis, improving and relieving symptoms and avoiding or adequately controlling recurrences.

Psychotherapy is a keystone in the treatment of functional gastrointestinal disorders. Not often, however, are the services of a psychiatrist necessary. Given, as needed, mild sedatives, certain forms of specific treatment in specific conditions, general measures and good hygiene and sympathetic understanding, the patient may be expected to recover or improve.

Too often the diagnosis of functional gastrointestinal disturbances is made on the basis of exclusion when evidence of organic disease is lacking. However, the diagnosis should be made on the basis of positive evidence, of symptoms characteristic of these disorders—manifestations just as characteristic of functional disturbances as are those of organic disease when the cause is organic change (Table 1). The diagnosis of a functional gastrointestinal disturbance should be suspected from the history given by the patient and confirmed by suitable physical examination and laboratory and roentgenologic studies. The diagnosis of these conditions is particularly difficult for physicians who cling to the old ideas concerning the origin of the symptoms previously discussed.

It has been said that if the physician will sit and listen, almost every patient will give enough information to permit the correct diagnosis to be made or at least suspected. Good listening is particularly important to the physician who deals with functional problems. Symptoms are sought which suggest familiar complexes and syndromes. As the story progresses questions are asked to complete a recognized pattern of either functional or organic disease.

Certain positive features strongly suggest a functional disturbance. These include symptoms that are characteristic and typical—for example, globus, aerophagia or functional vomiting. These features may be readily recognized in some cases, in others difficult to elicit. Other positive features include long continuance of symptoms without significant impairment of general health; failure to conform to the pattern of any recognizable organic disease; a bizarre timing of symptoms, such as the regular occurrence at six-weekly intervals, the close relationship of them to external stress and emotional disturbances; constancy of symptoms (particularly over a period of years); disappearance of symptoms during the night; the presence of pain with radiation in bizarre fashion to areas without established nervous system relationships; and the presence of burning pain, particularly when it is constant.

Associated Evidence of a Functional Disorder. The occurrence of gastrointestinal symptoms in a patient with other symptoms of an emotional disturbance should make one suspect the possibility of a functional gastrointestinal disorder. Among such associated symptoms are chronic fatigue and nervousness, anxiety and tension, being "tired all the time," having headache, insomnia, "light-headedness," numbness in the fingers and toes, difficulty in getting a deep breath, tremor and cardiovascular symptoms, including blushing, tachycardia, palpitation and cold, sweaty hands and feet.

It is always valuable to note whether the patient has had a normal emotional reaction to the symp-

TABLE 1. — *Points in recognition of functional gastrointestinal disorders.*

1. Positive features
2. Other evidences of functional disorders.
3. Various typical syndromes.
4. Lack of evidence of organic disease on examination.

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toms. Either undue concern or an unusually apathetic attitude toward the complaint should suggest to the physician that he may be dealing with a functional problem.

Various Typical Syndromes. A third significant diagnostic feature is the presence of a clinical syndrome typical of a functional disturbance, such as globus, aerophagia, cardiospasm, functional vomiting and hysterical bloating. However a functional disturbance may be present without causing such a typical syndrome.

Lack of Evidence of Organic Disease on Examination. The final point in diagnosis is the lack of evidence, on examination, of organic disease or of a disease that could produce the patient's symptoms. Physical examination must be thorough and complete. It frequently will reveal other evidences of functional disturbances. In addition to routine urinalysis and blood count, radiologic and laboratory studies should be carried out to the extent demanded by the judgment of the physician. It generally is wise to include studies of the organ which seems affected symptomatically or about which the patient expresses concern or doubt. For success in diagnosis as well as treatment, the patient and the physician must be satisfied that the studies have been complete and thorough.

It seems appropriate at this point to indicate the great importance of rectal and sigmoidoscopic examination, for these examinations all too frequently are overlooked or forgotten. It should be emphasized that occasionally examination of the stool for parasites and ova may give very useful information.

Accuracy of Diagnosis. The diagnosis of functional gastrointestinal disorders when made following adequate clinical evaluation is surprisingly accurate. Wilbur and Mills,[4] for example, some time ago carefully reexamined 354 patients who had a record of a previous diagnosis of functional gastrointestinal disturbance. The reexaminations were done at least seven years after the diagnosis was made and the original diagnosis was reaffirmed in 85 per cent of cases. The organic conditions most commonly found at the second examination which may have been missed and have led to symptoms at the time of the original examination were duodenal ulcer and gallbladder disease.

TREATMENT

The management of a patient with a functional gastrointestinal complaint is largely an art. In other words, it calls principally on the art of medicine in the treatment of a patient and not on the science of medicine in the treatment of a disease. For this reason quacks, charlatans, persons who treat with prayer or practice of religion and cultists without adequate training in the basic medical sciences are frequently highly successful in the management of patients with functional gastrointestinal disturbances. Too often the physician or surgeon skillfully trained in the science of disease approaches the patient from the standpoint of a disease or disturbed organ function and fails to visualize the disturbance in function of the patient as a whole. He does not recognize the "functional disturbance" presented by the patient and therefore is not successful in handling him or he lacks interest in doing so and passes him off as a "neurotic."

TABLE 2.—*Functional gastrointestinal disorders—Treatment.*

Art of Medicine vs. *Science of Medicine.*

Three steps to successful treatment:

1. Convince the patient of the diagnosis.
   (a) Accurate diagnosis.
   (b) Adequate explanation to the patient.
2. Improving or relieving symptoms.
   (a) Psychotherapy.
   (b) Diet.
   (c) Drugs.
   (d) General measures.
   (e) Psychiatric treatment.
   (f) Specific measures.
3. Preventing relapse.

Wide experience makes it quite clear that to be successful in management of a patient with a functional gastrointestinal complaint the physician must like the task just as a good surgeon must like to operate and a good obstetrician like to care for and deliver a pregnant woman.

As was previously mentioned, there are more hopeful avenues of approach to the management of "functional disorders" than is the case with most organic diseases. Briefly, the task of the physician who treats such patients is to establish the diagnosis, to reach the root of the difficulty and to work out an effective plan of treatment in the time he can afford to devote to a single patient. Sometimes this may be very simple, as in the case of a patient with anxiety over rectal bleeding which the patient suspects may be due to cancer of the rectum. But commonly the problem is not so simple and at times it may be very difficult.

The physician who is treating a patient for functional disorder must develop a plan tailored to fit the individual problem (Table 2). Three steps are essential. The first is a well thought out way to convince the patient of the diagnosis; second, a carefully considered program of specific therapy directed toward the patient, his conflict and his symptoms; and, finally, an attempt to show the patient how to remain largely symptom-free and to prevent relapses once improvement has occurred. The physician is guided in what he says and how he says it by his estimation of the patient's intelligence,

education, character, past experience and degree of sexual, psychological and philosophical maturity as well as by the characteristics of the conflict situation.

Convincing the patient of the diagnosis requires two steps. The first is an accurate diagnosis and the second an adequate and satisfactory explanation of the situation to the patient. Rare, nowadays, is the patient who is satisfied with the statement, "There is nothing the matter with you; it is 'just nervous indigestion.'" Especially in functional disorders the patient must be convinced of the diagnosis if treatment is to be successful. The physician certainly must let it be known that he does not look upon the symptoms as imaginary. At the same time the patient must be convinced that the symptoms are the body's "normal" response to prolonged anxiety or tension and not indicative of a severe or incurable disease.

It should be relatively easy to show a patient how stress and tension may result in gastrointestinal symptoms. Almost everyone has had some form of gastrointestinal symptoms under stress or with an emotional upset. The patient must be told that a certain amount of tension or stress is a normal part of every day life and that each person has a limit of tolerance for tension or stress and that when that limit is exceeded, symptoms occur. Every effort should be made to preserve the self respect of the patient and the feeling that he is respected in the eyes of members of his family and close friends. When the patient asks questions about the condition, the physician must have an answer to every question and the answers must be consistent.

Above all the patient must be given hope, for hope is the one thing all of us want when we are in trouble.

IMPROVING OR RELIEVING SYMPTOMS

Psychotherapy is the keystone in treatment directed toward relieving symptoms. As "specific therapy" it may be simple or it may require the services of a psychiatrist. Fortunately in most instances the general physician can successfully deal with such a patient. Whitehorn well expressed the situation: "Psychotherapy will consist largely in the thoughtful and respectful consideration with the patient of how the situation might be met more effectively, not by an ideal person, but by the person who is the patient, using to the best advantage the assets and attitudes which he has shown in periods of good adjustment. The whole art of psychotherapy depends largely on learning how to exert this special personal influence strategically to the patient's best advantage in finding a better way to meet a life situation." The establishment of rapport so good that the patient will tell the physician anything and look upon him as a knowing, sympathetic person, is extremely helpful.

Owing to an innate desire to know about themselves, most patients are helped by a free discussion of just what is wrong with them.

Diet. So variable are the needs that it is difficult to generalize about the dietetic management of patients with functional gastrointestinal disturbances. In general, however, a smooth diet, adequate in proteins, vitamins and calories and with as little restriction in variety as possible, best meets the average situation. An increase or decrease in calories depending on the need for a gain or loss in weight may bring about success in some patients. For patients with little appetite, or for hospitalized patients, feedings as far apart as possible (at least five hours) usually are helpful. The need for reduction in roughage or an increase in it when diarrhea or constipation occurs is obvious. Patients should be encouraged to eat slowly and not to eat when tense or under too much stress. At times omission of coffee, alcohol or tobacco may be very helpful. Finally, for patients who require much dietary consideration the services of a skillful and sensible dietitian are invaluable in working out the details of a program.

Drugs. Sedatives are the most useful drugs in the treatment of functional gastrointestinal disturbances. They are not curative but, like a crutch, are exceedingly useful in getting the patient over a crisis, in helping him to regain his feeling of well-being and in giving him an insight into what it feels like to be eased or free of symptoms. Small doses of barbiturates and the occasional intermittent use of chloral hydrate or bromide, prescribed at times in a form new to the patient, and used during the day and to obtain sleep at night, may change the whole outlook of a nervous patient with gastrointestinal symptoms. It is wise to limit the amount of drugs and the period over which they are to be used. In the author's experience not many patients, except the severely psychoneurotic, become psychologically dependent on barbiturates. As physicians feel no grave concern over the way the average "normal" person drugs himself daily with caffeine, nicotine, alcohol and often acetylsalycilic acid, the occasional addition of barbiturates to this daily ration should cause no serious misgivings.

Next to sedatives, antispasmodics in the form of belladonna and its alkaloids and of the many recently developed synthetic anticholinergic agents are the most useful drugs in therapy of functional disturbance. Curiously, a particular anticholinergic drug may work exceedingly well in one patient when others do not, whereas in another patient the converse may be true. Because belladonna and atropine are so commonly satisfactory and so much less expensive, they should be used first.

Chlorpromazine, which acts as a mild depressant to the central nervous system, may be very helpful in controlling functional nausea and vomiting and in augmenting the effects of other drugs in the management of abdominal discomfort and pain.

Antacids, vitamins and dilute hydrochloric acid properly used may be helpful. In some cases smooth bulk substances are helpful in the control of constipation and occasionally in diarrhea; and a variety of symptomatic measures for the control of diarrhea, such as administration of kaolin, bismuth, pectin and even small doses of opiates, may be extremely useful in handling an acute situation or bringing a chronic one more satisfactorily under control. The proper control of chronic constipation alone is one of the most helpful means of relieving functional gastrointestinal disturbances.

Vitamins, particularly of the vitamin B complex, frequently are very helpful in patients who have been on limited diets; in others they have a useful tonic or psychologic effect.

Any discussion of drugs in treatment of patients with functional gastrointestinal disturbances should include a consideration of the placebo response so well emphasized by Wolf. Improvement that follows administration of a particular drug or group of drugs may be wholly psychic although the good effect may be attributed by both physician and patient to the pharmacologic effect.

Recent observations of Beecher and his associates have further advanced understanding of the phenomenon of the placebo response. In study of 162 postoperative patients observed for ability to obtain significant relief of pain from subcutaneous injections of morphine and placebos, these observers found a considerable number of patients who responded with relief of pain following injections of placebos. Fewer than half the patients who received multiple doses of placebos responded consistently to them. There were no sex differences and no differences in intelligence between placebo reactors and nonreactors. In general placebo reactors in stressful postoperative situations behaved in immature, dependent and more outwardly responsive fashion and "thus receive considerable relief of pain through comfort received from attentive nursing care and from confidence in the effectiveness of drugs," while the nonreactor, "withdrawn and rigidly clinging to critical intellectual processes, is less comforted by the care received and evidently more critical of drug effects." Placebo reactors were more productive of response, more anxious, more self centered and preoccupied with internal bodily processes and more emotionally labile.

Much more study and observation must be carried out in this important field before clinicians can properly evaluate the nature of response of patients with functional gastrointestinal disorders.

General Measures. Rest and exercise, extroverting activities, the development of suitable outlets and hobbies and the taking of vacations may be most helpful in the long-time planning of treatment of chronic functional gastrointestinal disturbances.

Specific Measures of Treatment. There are certain specific measures of treatment useful in specific types of functional gastrointestinal disease. These include dilatation of the esophagus for spasm, a dry diet in functional vomiting and aerophagia, voluntary control of belching in aerophagia, manual dilatation of the tight anus in cases of constipation and irritable colon, and the use of smooth bulk substances and abdominal exercises in constipation. An elimination diet may be extremely helpful in patients who suspect they are allergic or who insist that certain foods cause distress, but trial of a limited diet should be restricted to short periods.

Psychiatric Treatment. General physicians can properly treat most patients with functional gastrointestinal disorders. Occasionally in the case of a patient who has severe chronic psychoneurosis or who has psychotic symptoms such as those of depression, or who reflects a notable absence of emotional concern about his condition, or is hysterical or has conflicts which involve deep guilt or serious disturbance of one of the basic emotional drives, a competent psychiatrist should take over the case. Sometimes, however, a physician may carry such a patient along at a superficial level until he is willing to see a psychiatrist.

PREVENTION OF RECURRENCE

Successful management of patients with functional gastrointestinal disorders does not end with control of the presenting situation. Many patients have recurrent bouts of tension, anxiety and gastrointestinal symptoms. Such a patient should be told to expect symptoms whenever tolerance for tension is exceeded. He may avoid a situation that will result in anxiety and tension. Through treatment he may develop insight into his symptoms and build up stress tolerance to the maximum. He may be taught to avoid long and needless worry about new and persisting symptoms and he may also learn simple dietary and therapeutic measures for handling recurrence of symptoms. Since his problem is a medical one, he should not hesitate to seek the advice of his physician at regular intervals or when he gets into difficulties that he cannot himself resolve.

PROGNOSIS

The prognosis of patients with functional gastrointestinal disturbances varies greatly and may be

very difficult to estimate. In general the prognosis for patients with simple anxiety and tension and fatigue states is good, particularly when anxiety can be relieved and fatiguing work restricted and rest prolonged. Young persons with emotional and environmental problems that can be solved often improve remarkably and recover completely. For patients with pronounced chronic emotional disturbances or prolonged economic or environmental conflicts that cannot be altered or to which they cannot become accustomed, and for patients with psychotic disturbances, the outlook is often not too good; yet the patient often can be improved, and even a little improvement may be enough to make life worth living again.

ADDENDUM

After careful consideration of this whole problem one cannot but agree in part at least with Josh Billings, who said, "I have finally kum tu the konklusion that a good reliable sett ov bowels iz wurth more tu a man than enny quantity ov brains."

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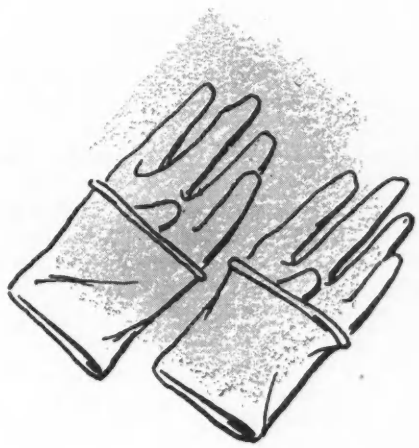

Surgical Treatment of Bleeding Esophageal Varices

ROY COHN, M.D., San Francisco

• *Massive bleeding from varices in the esophagus and stomach secondary to liver disease is a serious surgical emergency, as the patient may bleed to death. The problem is further complicated by the difficulties in making a diagnosis and the poor general condition of the patient due to the long standing liver disease. For the past two years at the San Francisco Hospital this problem has been handled by exploring the stomach and esophagus and ligating the bleeding point. No effort has been made to lower the pressure in the veins to the liver. The results have been sufficiently encouraging to warrant further trial.*

MASSIVE BLEEDING from the gastrointestinal tract secondary to liver disease is a serious problem that is faced relatively often in large city hospitals. The various forms of surgical treatment now in use to stop bleeding of this kind are: Ligation of splenic artery; ligation of hepatic and splenic arteries; injection of varices with sclerosing solutions; esophagogastrectomy and total gastrectomy; esophageal and gastroesophageal tamponade; and ligation of varices transthoracically. Varying degrees of success have been reported from each of the methods. It is the purpose of this presentation to report experiences in 12 cases in which an attempt was made to control the bleeding during the hemorrhage by transthoracic ligation of the bleeding point.

Direct surgical attack on bleeding varices has been described by Boerema (cited by Linton and Warren[7]), Crile[5] and Linton and Warren.[7]

The surgical problem is complicated by two important factors: the poor condition of the patients with consequent high fatality rate from the hemorrhage, and the uncertainty of the diagnosis in many instances. The diagnosis of esophageal varices is by no means certain unless one is fortunate enough to have had previous gastrointestinal or esophagoscopic examination of the patient. The presence of cirrhosis of the liver, while helpful, does not rule out an ulcer of the stomach or duodenum as a source of the hemorrhage. At autopsy of 68 patients with hepatic cirrhosis who died from hemorrhage at the San Francisco Hospital, bleeding was shown to have come from a duodenal ulcer in four patients. This well known association of cirrhosis and ulcer was most recently pointed out by Fainer and Halsted.[6] Twenty-nine per cent of the patients they reported upon had two clinically demonstrable lesions.

Esophageal varices cannot be demonstrated in all patients with cirrhosis who bleed. In the previously mentioned autopsy series, 23 of the 68 patients had no demonstrable cause of bleeding. Conversely, esophageal varices can occur in patients who do not have cirrhosis. Failure to demonstrate varices may be due to the fact, as Palmer and Brick[8] have shown, that autopsy and x-ray techniques are too crude. They were able to demonstrate varices by esophagoscopic means in 22 patients who did not have cirrhosis of the liver. It is interesting that in a few of their cases in which the portal pressure was measured through the esophagoscope, the measurements were within the normal pressure range.

In an attempt to solve this diagnostic problem, some clinicians have utilized the Sengstaken tube.[10] With the balloon in place, material aspirated from the stomach would show blood in patients with bleeding ulcer. The author's experience with the use of the Sengstaken tube is limited, but it has been found to be difficult to place and difficult to hold in place. Moreover, blood was aspirated in some cases even when the bleeding was proved to be from varices. One patient vomited up the balloon in the distended state, and in two patients bleeding began again the moment the pressure was released.

Although the author has not had much experience with gastroesophageal barium examination in the presence of massive hemorrhage, it is difficult to see how a roentgenologist can do an adequate examination on a patient bordering on shock whose stomach may be filled with clots.

Because of this difficulty in accurate diagnosis of the source of hemorrhage, it is felt that in every case in which operation for control of bleeding is done, the stomach ought to be opened. The most adequate approach to the stomach and lower esophagus, as well as the best exposure if a splenorenal anastomosis is necessary, is through a left thoracoabdominal incision.

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In the case of a few patients with atypical history in the present series, 75 cc. of 35 per cent Diodrast® was injected into the spleen. This is a simple procedure, and the x-ray film taken at that time may demonstrate an extrahepatic obstruction (as shown in Figure 2), the only indication for a decompression operation in addition to ligation of the bleeding point.

It is important from the surgical point of view that the patients with bleeding are likely to be in extremely poor state of nutrition and in many instances cannot live long if bleeding continues. This is true generally of patients with cirrhosis. The onset of the hemorrhage in many of these patients seems to occur after a long bout of alcoholism.

Table 1 gives data on the patients in the present series who were operated upon with a presumptive diagnosis of hemorrhage from esophageal varices. It is of interest that in three patients the bleeding point was from a gastric varix. In one of these patients the gastric varix was in a hiatus hernia. There were two cases of extrahepatic obstruction, one certain and one questionable. There were two deaths. One patient died during induction of anesthesia from vomiting and aspiration. Thereafter in all cases the intratracheal tube was placed under local anesthesia before the operation was started. The second death occurred on the twelfth postoperative day and was due to leakage of the gastrotomy suture line with mediastinitis in a severely cirrhotic patient who also had delirium tremens postoperatively.

A left thoracoabdominal incision is used for best exposure of the lower esophagus and stomach. At operation the spleen usually is removed first to facilitate exposure. The splenic vein is preserved for possible anastomosis. The pressure is measured

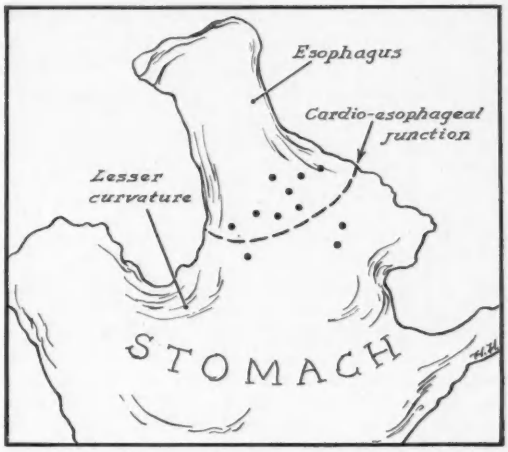

Figure 1.—Diagram showing source of hemorrhage in those cases where a single point was found to be bleeding.

directly in the portal system and the stomach is opened high, near the esophageal junction. If no evidence of ulcer is found in the stomach or duodenum, the esophageal hiatus is exposed with anal retractors. If hemorrhage is in progress, locating the source of bleeding is not difficult, but it may not be from a single point. If the bleeding varix can be discovered, it is sutured with a running stitch.

Curiously, although in many cases varices extend the entire length of the esophagus, the bleeding seems to occur invariably either in the stomach or esophagus, within about 5 cm. of the cardioesophageal junction. While it is possible that esophagitis is the cause of hemorrhage in cases in which the bleeding comes from an esophageal varix, it certainly does not explain the bleeding from gastric varices. Undoubtedly there are many causes for the

TABLE 1.—*Data on 12 cases in which transthoracic operation was done to stop bleeding from esophageal varices*

Patient	Age	Sex	Diagnosis	Bleeding After Suture	Deaths	Follow-up (Months)
1	54	M	Cirrhosis	0	0	11
2	61	M	Cirrhosis, Gastric varix.	0	0	14
3	44	M	Cirrhosis	Yes	0	6
4	9	F	Cirrhosis (PCA 1952)	0	Yes	Cholemia 6
5	28	M	Extrahepatic obstruction	No bleeding source demonstrable	0	6
6	38	M	Cirrhosis, Gastric varix	0	0	9
7	65	M	Cirrhosis	0	0	14
8	64	M	Banti's syndrome ? Marginal ulcer ? Extrahepatic obstruction	Yes	0	2
9	68	M	Cirrhosis	0	0	18
10	62	M	Cirrhosis	0	0	5
11	50	M	Cirrhosis	0	Yes	12 days
12	62	F	Cirrhosis	Yes	0	11
13	47	F	Cirrhosis	No bleeding source demonstrable 0	Yes	Died during induction of anesthesia

hemorrhage in such cases. One patient was observed in whom hemorrhages occurred twice in small amounts after a ligation operation, and both times the bleeding followed a severe bout of coughing.

Figure 1 is a diagram illustrating the point of obstruction in the 11 cases in which a single source of bleeding was discovered.

Following are reports of illustrative cases.

CASE 1. *Extrahepatic obstruction, proven by x-ray; splenorenal anastomosis.*

An 18-year-old white man was sent to the hospital on January 20 because of gastrointestinal bleeding. The patient had been well until January 18, when he reported to the surgical clinic for evaluation of an inguinal hernia. At that time an indirect hernia was felt on the right side. The patient stated that he had had an enlarged spleen since the age of seven, but he did not want operation for the spleen since, he said, it had been taken care of by previous treatment. (The spleen could be felt on deep palpation.) The patient left the surgical clinic and that night felt weak and defecated black stools. The following day he again felt weak and had a black stool. On the morning of January 20 he became nauseated and vomited about four times. The first vomitus was dark brown in color and subsequent vomitus contained bright red blood. The hemoglobin content at that time was 7.5 gm. per 100 cc. of blood. Two days before it had been 10 gm.

The patient had had the first episode of hematemesis and melena at age 7, when he was kicked in the stomach by a playmate. A second episode occurred at age 8 when he did some heavy lifting. At age 12 and 15 he again had episodes of bleeding. The enlarged spleen first was noted on physical examination at age 12.

The patient was well developed and well nourished but pale. The spleen was palpable four finger-breadths below the costal margin. It was firm and slightly tender. The liver and other organs were not palpable. The blood pressure was 130/60 mm. of mercury and the pulse rate 100. An indirect inguinal hernia was present on the right side.

On January 20 and 21 the patient was given six blood transfusions of 500 cc. each and the hemoglobin content rose to 11.9 gm. per 100 cc. Esophagoscopy was done on the afternoon of January 20 and varices were seen, but no actual bleeding point was demonstrated. The patient vomited several hundred cubic centimeters of dark reddish-brown material during the procedure. A Sengstaken tube was inserted. On January 22, following the transfusions as noted above, the patient was doing quite well and the esophageal balloon was deflated and old blood returned from the stomach. The patient was given another 500 cc. of blood at that time. The esophageal balloon was reinflated. On January 24, with bleeding continuing, another 1,500 cc. of blood was required and, accordingly, surgical treatment was instituted. A preoperative hepatogram was made (Figure 2).

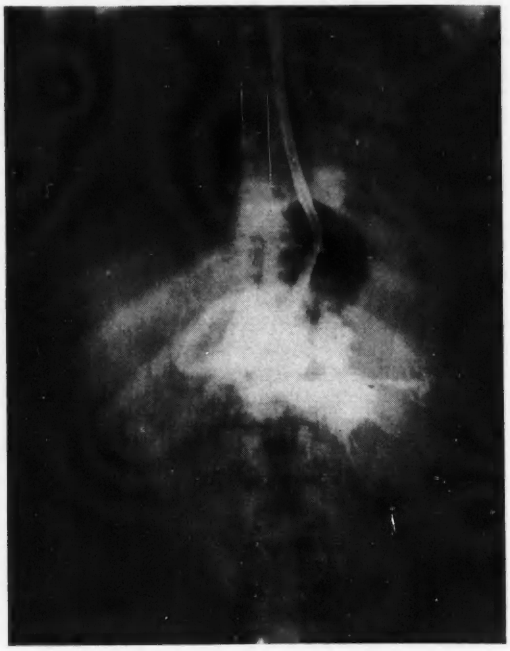

Figure 2.—The hepatogram in Case 1, showing no dye in the liver. The spleen is outlined. Interpretation: Complete extrahepatic obstruction. Operation: Splenorenal anastomosis.

The chest and abdomen were opened through a combined thoracoabdominal incision through the eighth interspace, the diaphragm was opened and the spleen was exposed. The spleen was approximately five times normal size. Splenectomy was carried out, the splenic vein being preserved. This vein was not thrombosed. The stomach was opened on the anterior surface and very large varices were observed. No attempt at ligation was made as no one bleeding point could be seen, and the stomach was closed. The pressure, measured in a tributary of the splenic vein, was 345 mm. of water. Splenorenal anastomosis was carried out. Following this procedure the pressure, determined in one of the tributaries of the splenic vein, was 210 mm. of water. The liver was normal in appearance and was not enlarged. The patient made an excellent postoperative recovery. The Levine tube was removed four days postoperatively. The hemoglobin content following operation was 13.1 gm. per 100 cc. and did not again decline. The sutures were removed on the seventh postoperative day and the wound was well-healed.

CASE 2. *Hemorrhages from a gastric varix within a hiatus hernia in a cirrhotic.*

A 50-year-old white man was admitted to the hospital on February 9. He had been vomiting blood every hour since awakening on the morning of entry. The week before entry the patient noted black stools several times daily.

The patient had been admitted to hospital thrice previously in the preceding four years for Laennec's cirrhosis, alcoholism, ascites, edema, avitaminosis and bleeding esophageal varices.

When examined the patient was sitting up in bed. Slight flaccidity of the face and gross tremor of the arms were noted. There were vascular spiders over the nose, left cheek and anterior chest and increased venous markings of the right anterior chest and right abdomen.

The patient continued to vomit blood and had tarry stools. He went into shock with blood pressure at 66/44 mm. of mercury on the evening of entry, and after two units of blood had been given he was taken to surgery. Through a left thoracoabdominal incision gastrotomy was performed and several actively bleeding varices were ligated with gut sutures in the region of the cardia of the stomach. There was believed to be a small hiatal hernia present also. No ulcers were noted. The spleen was removed. A specimen of the liver was taken for biopsy. The patient received six units of blood during operation.

Postoperatively, the patient had fever, subcutaneous emphysema and purulent exudate from the chest wound. Thoracentesis was carried out February 16 and 65 cc. of serosanguinous material was removed. It contained Gram-positive streptococci and staphylococci. Cultures were not reported. On February 18 dehiscence of the thoracic wound developed and a sucking sound could be heard. Left lower lobe pneumonia with three fluid levels and definite empyema were noted in an x-ray film of the chest. Penicillin, streptomycin and supportive intravenous therapy were given but the patient died on February 21.

At autopsy a leak was noted at the suture line in the gastrotomy wound and mediastinitis was present. The bleeding varix, which was thrombosed, was located in the gastric side within a small esophageal hiatal hernia. The liver was cirrhotic.

CASE 3. *Hemorrhage from a gastric varix. Mild cirrhosis.*

A 64-year-old male Negro prisoner entered the hospital in November 1953 because of massive hematemesis of a few hours' duration. At a previous entry a diagnosis of suspected cirrhosis of the liver had been made. No abnormalities were noted in a gastrointestinal series done at that time.

A presumptive diagnosis of bleeding ulcer was made and operation was done. Only slight pathologic changes were noted on biopsy of the liver. When the stomach was opened a small fountain of blood was seen pouring from one of several gastric varices. The varix was stitch ligatured.

After recovery the patient was sent back to prison, where he has had no further hemorrhage in 14 months.

CASE 4. *Repeated hemorrhages from varices. Cause undetermined.*

A 59-year-old white man entered the hospital in February 1955 for the ninth time with gastrointestinal bleeding. He had had this condition intermittently since 1947. In 1948 a gastric resection was done at another hospital, but no ulcer had been demonstrated. The patient first entered San Francisco Hospital in 1951. No blood dyscrasia had ever been demonstrated, although a diagnosis of erythroblastic anemia had been suggested. The spleen was large and esophageal varices had been demonstrated roentgenographically. On the eighth entry in September 1954 for severe hemorrhage, operation was performed. The liver did not appear grossly diseased. Portal pressures were within normal limits. The spleen was removed and the stomach opened. No marginal ulceration was found. There was some oozing from some small veins in the distal esophagus. These were stitched. In February 1955, after a drinking bout, the patient again vomited some blood, but on supportive therapy the hemorrhage stopped.

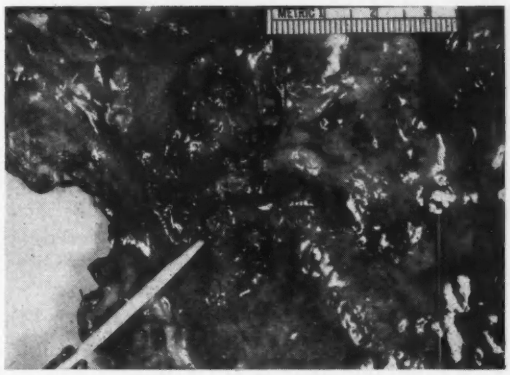

Figure 3.—The thrombosed varix in Case 2. The bleeding point was within a hiatal hernia. The sutured varix can be seen parallel to the gastroesophageal junction.

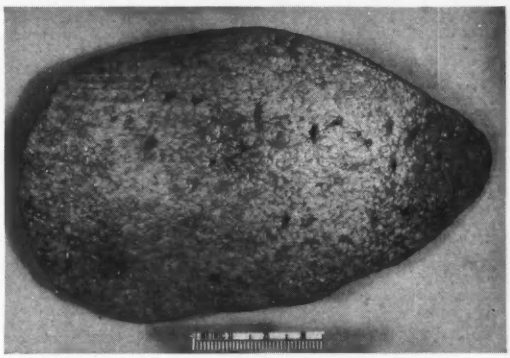

Figure 4.—The appearance of the liver in Case 2.

DISCUSSION

The small series of cases reported would suggest that direct attack on bleeding esophageal varices is feasible. There does not seem to be any doubt about

this fact in the case of the patient with extrahepatic portal obstruction as the cause of esophageal varices. In these patients, splenorenal anastomosis should be done at the time of operation.

In the case of patients with cirrhosis of the liver, the problem is not quite so clear cut. It is obvious that ligation of one varix where several are present hardly can be expected to control the bleeding more than temporarily. Linton and Warren expressed the opinion that a portacaval anastomosis should be done after ligation, at some time in the postoperative period when the patient's condition has improved.

The author has not done portacaval anastomosis, for it is felt that lowering of the blood flow to the liver in the presence of cirrhosis is as damaging in these poor risk patients as any possible theoretical lowering of the pressure in the varices may be in the prevention of further hemorrhage. It is therefore of interest to note that the surviving patients in the series here reported, who have not had any form of portal decompression, seem to have about the same results as those reported by Linton and Warren who had portal decompression.

ADDENDUM

Since the presentation of this report, three of the surviving patients have bled again in small amounts. In one patient with large varices demonstrable by x-ray, portacaval anastomosis has been done.

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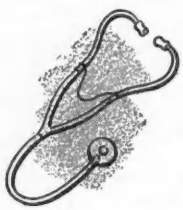

Needle Biopsy of the Kidneys

Studies of Two Cases of Lower Nephron Nephrosis Due to Toxic Solvents

WILLIAM C. CASEY, M.D. and WILFORD WOOLF, M.D., TORRANCE

• *In two cases of "lower nephron nephrosis" or acute renal failure, needle biopsies of the kidney were performed. The first case developed in a 46-year-old woman following inhalation of vapors from an insecticide spray and a cleaning fluid. The second case was due to ingestion of carbon tetrachloride.*

Histologic study of the human kidney during toxic nephrosis showed changes confined mainly to the epithelium of the proximal tubules, confirmed the diagnosis, and illuminated the clinical course.

Needle biopsy of the kidney during acute renal failure did not influence the course of the disease unfavorably. It was easy, and entailed no complications.

Needle biopsy of kidneys may prove as informative and valuable as needle biopsies of the liver by aiding in the prognosis as well as in the diagnosis of various renal diseases.

THE PRESENT REPORT is an analysis of findings in the study of needle biopsies of the kidneys in two patients with acute toxic nephrosis probably caused by organic solvents. The specimens illuminated the course of the disease and removing them did not seem detrimental to the patient. A correlation of histologic with clinical and laboratory changes was made. More experience with similar instances may make evaluation easier.

Needle biopsy of the kidney was first reported in 1951,[3] and subsequently was shown to be a safe and valuable adjunct in the study of renal diseases.[6,7] Apparently few complications other than microscopic hematuria have been caused by the procedure, and the authors' experience with 12 patients corroborates this conclusion. Renal tissue is not always obtained in the needle; fat, connective tissue, muscle, and in one instance, liver tissue have been found. Despite the relative inaccuracy of the present technique, renal tissue is obtained frequently enough (in about 60 per cent of attempts) to warrant its continued use.

Before using this procedure in humans, caution dictated trial in animals. During the course of unpublished experiments on the renal circulation in guinea pigs, the Turkel needle for biopsies was used at operation to secure cores of renal tissue, and subsequent observation showed minimal bleeding. Later autopsies at varying intervals showed no serious damage to the kidneys.

These results encouraged the authors to perform needle biopsy of the kidney in patients with various renal diseases. Attention was directed early to the various forms of acute renal failure. As Swann and Merrill[8] pointed out in their exhaustive review on acute renal failure, anuria may occur in a variety of widely separated conditions, such as heat strokes, cholera, alkalosis and vomiting, trauma hemorrhage, intravascular hemolysis, shock, the crush syndrome, severe burns, and exposure to chemical nephrotoxins.

Oliver[5] postulated two common factors in the production of acute renal failure—renal tubular poisoning, and severe ischemia. These factors seem to adequately explain the clinical picture and the anatomic lesions. So far as could be determined there are no reports of serial biopsies of the kidney during the course of this disorder. All pathologic lesions described to date were seen either in experimental animals or in human kidneys obtained at autopsy. Considering how the use of needle biopsies of the liver has enriched the knowledge of hepatic disorders, an application of this procedure to certain renal diseases seemed warranted.

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Submitted February 8, 1955.

REPORTS OF CASES

CASE 1. A 46-year-old white woman was admitted to the hospital on October 14, 1953, with complaints of upper abdominal pain, backache, nausea and vomiting and fever. The patient had been well until four days previously when, having cleaned curtains with cleaning fluid* and sprayed plants in the garden with insecticide,† she felt weak, became nauseated, and collapsed. She then vomited, noticed a backache, and had fever. Four days later she sought relief at the hospital.

*Chemical analysis of this material showed the presence of petroleum naphthas with some unidentified impurities. No carbon tetrachloride or benzenes were found.

†This insecticide contained the following chemicals: DDT 5 per cent, chloranane 0.03 per cent, betabutoxybutane 0.65 per cent; pyrethrum 0.04 per cent, ethanolbutoxide 0.85 per cent, methylated naphthalenes 10 per cent; essential oils 0.04 per cent, and petroleum distillates 83.06 per cent. (Taken from manufacturer's label.)

At the time of admittance the blood pressure was 140/100 mm. of mercury, the temperature 97.4° F., and the pulse rate 107. The abdomen was slightly distended, and the liver was palpable two finger-breadths below the costal margin. The abdominal muscles were voluntarily contracted, and bilateral flank tenderness was present. A plain roentgenogram of the abdomen showed distention of the small and large bowel. The hemoglobin content of the blood was 14.1 gm. per 100 cc. The hematocrit was 46 per cent. Leukocytes numbered 14,100 per cu. mm. The blood urea nitrogen was 30 mg. per 100 cc. There were many leukocytes in the urine.

The patient was thought to have either acute gastroenteritis or obstruction of the small bowel and was managed accordingly. However, on the sixth day of illness, only 50 cc. urine, which was bloody, was passed in 12 hours. Cystoscopic examination and ureteral catheterization showed no urinary tract obstruction, and retrograde pyelograms were normal. Lavage of both renal pelves with sterile saline solution was carried out and a small amount of dark brown liquid was obtained. Microscopically, a few erythrocytes and many large red crystals which were thought to be heme pigment were seen.

A diagnosis of acute renal failure was made, and a needle biopsy of the right kidney was uneventfully performed approximately 12 hours after the onset of oliguria.

The patient was then treated on the basis of principles reported by Bull and associates.[1] She remained oliguric for 12 days; nausea, vomiting, and diarrhea persisted. The blood urea nitrogen and creatinine rose to high levels. Diuresis began after 12 days of oliguria, and progressed through polyuria to a normal urinary output. Another specimen was obtained for biopsy (from the left kidney) after the start of diuresis, and a third biopsy was performed (on the right kidney) at about the height of the polyuric phase. The patient made uneventful recovery, and when seen four months later was well.

DISCUSSION

When oliguria was first detected, obstructive urinary disease was excluded by cystoscopic examination and retrograde pyelography (as indicated by Harrison[2]). Corroboratory evidence of acute renal failure was obtained by the return of heme crystals through the ureteral catheters after gentle lavage of the renal pelves. The six-day delay in onset of oliguria was greater than is usual for patients exposed to nephrotoxic chemicals.

To maintain fluid balance, fluid intake was restricted immediately to about 800 cc. per day plus a quantity equal to the estimated loss from urine, vomitus and stool. Intravenous administration was necessary since nausea and vomiting were present. Thus, overhydration and subsequent pulmonary edema and cardiac failure were avoided in the oliguric phase (Chart 1).

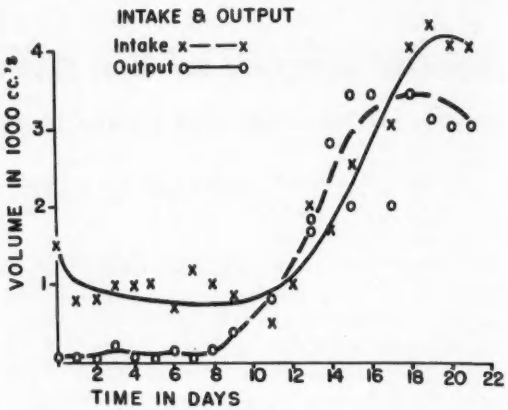

Chart 1 (Case 1).—Urinary output compared to total fluid intake. Fluids should be limited to 800 cc. more than the urine volume per 24 hours.

In polyuria, fluid balance involved replacement not only of the insensible loss and loss through vomiting and diarrhea, but also of water and electrolytes lost in the large volume of urine excreted by the recovering kidneys.

It was not until the twelfth day of the diuretic phase that nausea and vomiting ceased. Shortly thereafter the patient was able to take an adequate amount of liquids and intravenous administration was stopped.

The persistent nausea, vomiting, and diarrhea increased the problem of fluid and electrolyte management. Careful and frequent determination of the serum sodium content showed low values, around 130 mEq. per liter, throughout most of the illness. The chlorides measured between 75 and 90 mEq. per liter until the patient was well into the polyuric phase. Little sodium was lost in the urine in the oliguric phase and replacement was limited to the estimated loss from vomiting and diarrhea. Hypernatremia, which can produce not only pulmonary edema but also an edema of the kidney which would prolong the recovery period, was thus avoided. Chlorides were administered with the sodium in equivalent quantities. (Ordinarily in the oliguric phase water and glucose suffice, but in this patient the loss through the alimentary canal necessitated replacement.)

In the polyuric phase excess electrolyte and water loss occurred. It is believed that the regenerating tubular epithelium is functionally immature, and therefore unable to conserve essential elements for the body. The loss of sodium was estimated at approximately 100 mEq. per liter of urine. Replacement each day was guided by the previous day's losses, with one exception. Early in the diuretic phase a quantity of 5 per cent sodium chloride was administered intravenously in anticipation of

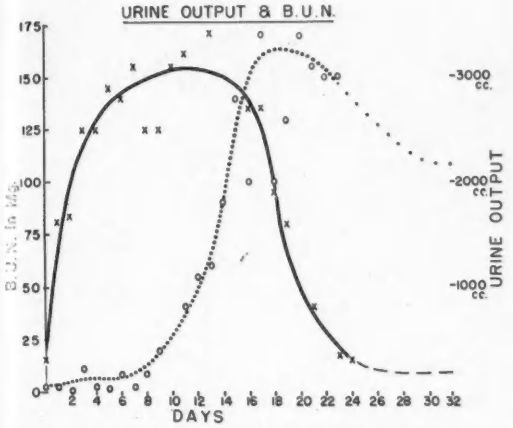

Chart 2 (Case 1).—Urinary output (dotted line) compared with blood urea nitrogen (solid line). As the urine output increased, the blood urea nitrogen (BUN) fell rapidly.

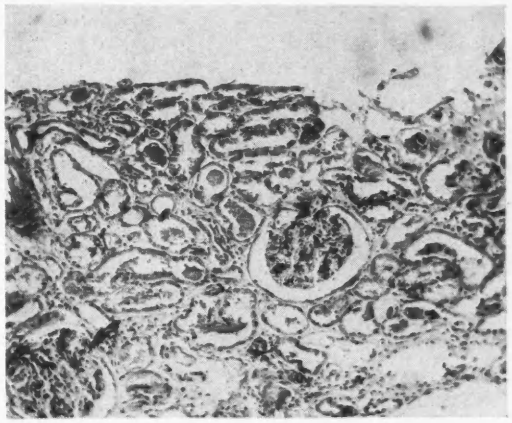

Figure 1 (Case 1).—Biopsy specimen (low power) on first day of anuria. Note necrosis and dilation of some of the tubules. Protein material is evident in many of the dilated tubules. The interstitium appears normal.

polyuria with its accompanying large losses of electrolytes. This decision proved to be an error in judgment, as acute pulmonary edema, which fortunately rapidly disappeared on digitalization and temporary restriction of salt, promptly developed. This experience provided a vivid illustration of the hazards of excess sodium in oliguria.

The potassium ion levels in the serum were normal in the oliguric phase. This is of some interest, as hyperkalemia usually occurs. Possibly hyperkalemia was prevented by the large losses of potassium through the gastrointestinal tract. Since this is a serious and sometimes fatal complication of oliguria, the possibility of reduction of serum potassium by artificially-induced diarrhea (perhaps by giving drugs) is intriguing and further pursuance of this question would be interesting.

In the polyuric phase the decrease of serum potassium was readily corrected by intravenous replacement. Since the kidney has little ability to conserve potassium even in homeostasis, excess loss occurred in the typically large volume of urine excreted in the polyuric phase.

The blood urea nitrogen initially rose rapidly, and became normal after polyuria occurred (Chart 2). There was also a corresponding rise and fall of serum creatinine.

The usual anemia associated with acute renal failure also developed. The hemoglobin dropped from 14.4 gm. per 100 cc. on admission to 7.4 gm. by the twenty-first day. Much investigation has been done regarding possible causes of this anemia. In a recent study on bilaterally nephrectomized rabbits, hemolysis was emphasized as the primary causative factor.[4] The concept of depression of erythropoiesis in renal insufficiency likewise has its proponents. It is possible that both mechanisms are factors in the production of the anemia of acute renal failure. However, since the anemia is usually well tolerated, it is believed that blood transfusions are not indicated unless critically low hemoglobin levels are reached late in the disease. In agreement with others, the authors fear the effects of increased extracellular volume in patients who are oliguric. Also, it has been demonstrated that transfused erythrocytes die and liberate into the serum of the recipient large quantities of potassium, which are undesirable in the oliguric phase. If blood is absolutely necessary, small quantities of packed red blood cells should be administered after diuresis has begun.

Correlation of pathologic changes with clinical and laboratory findings. Since acute renal failure seems to be divided into two phases, it was thought that securing specimens for biopsy at the beginning and end of oliguria and during the height of polyuria would provide the most information with minimum risk to the patient. Biopsy was performed on only one kidney at a time.

Biopsy on first day of oliguria. Immediately after it became known that obstruction was not the cause of oliguria in the present case, a biopsy specimen of the right kidney was obtained which represented the structure of the right kidney 12 hours after functional failure. The section was of renal cortex (Figure 1). The overall architecture was normal. Several glomeruli were present, one showing slight hyaline thickening of Bowman's capsule. Glomerular capillaries showed slight hyaline thickening of the basement membranes, but were not totally constricted as capillary lumens were clearly demonstrable. Scattered red cells were noted in the glomerular capillaries. The proximal convo-

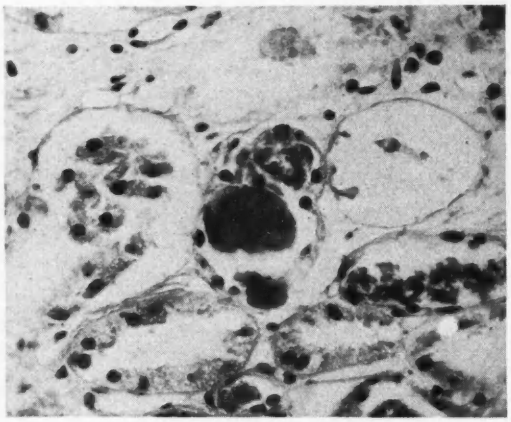

Figure 2 (Case 1).—Biopsy specimen (high power) on first day of anuria. A heme pigment cast in a distal tubule.

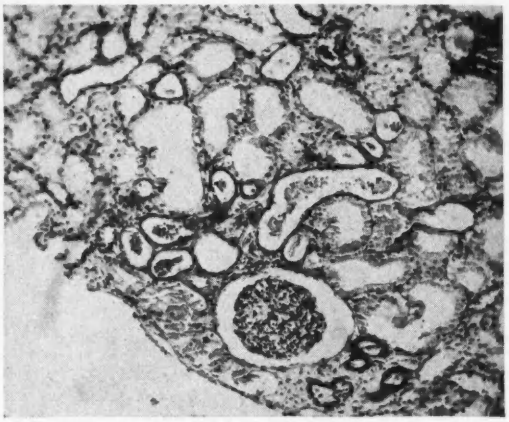

Figure 3 (Case 1).—Biopsy specimen (low power) at onset of diuresis. Dilation and necrosis of tubules still present, but some signs of regeneration evident.

luted tubules were moderately dilated and the epithelial lining cells were frayed and focally sloughed into the tubule lumens. Occasional tubules were partially devoid of epithelial lining. The distal tubules showed less epithelial sloughing. The tubule lumens contained many well-preserved erythrocytes (possibly related to the biopsy procedure). Cellular debris admixed with amorphous granular material with the staining characteristics of hemoglobin was present in both proximal and distal tubules (Figure 2). The arterioles were slightly thickened. No inflammatory cells were seen, and no tubulovenous communications were identified.

A question that seemed pertinent was: How do these anatomic changes relate to the clinical picture of extreme oliguria?

As Oliver suggested, the failure to produce normal amounts of urine may be caused by a reduction in renal blood flow and glomerular filtration. (In the previously mentioned biopsy specimen in the present case, only scattered red blood cells were seen in the glomeruli.) In addition, both obstruction of the tubules by heme pigment casts and tubular rupture undoubtedly contribute to diminished urinary output. The proteinuria may come from the sloughed epithelium and pink-staining protein debris in the tubules. The elevation in blood urea nitrogen and creatinine is explained by failure to excrete these substances, since little or no urine is eliminated. The combined factors of tubular damage and focal ischemia thus seem to explain the clinical picture.

Biopsy immediately after diuresis began. The patient eliminated 1,800 cc. of urine in the 24 hours preceding the biopsy of the left kidney. Surprisingly, at biopsy the kidney did not appear much changed. Despite extreme tubular damage (Figure 3), some evidence of tubular regeneration and reconstruction was visible. Many tubules were still remarkably dilated. In one section a group of tubules showed epithelium with extremely clear cytoplasm and centripetal displacement of the nucleus (Figure 4). This condition of hydropic degeneration of tubular cells was produced experimentally in animals by intravenous injection of sucrose (Bell). Perhaps the intravenous glucose administered to the patient was partially responsible for this lesion.

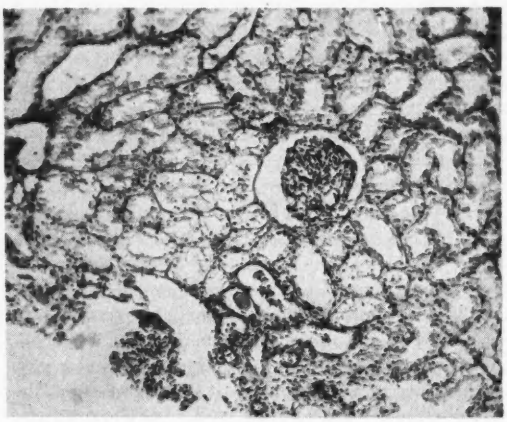

Figure 4 (Case 1).—Biopsy specimen (low power) at onset of diuresis. Many tubules near the glomerulus show hydropic changes. This may be due to the administration of intravenous glucose solution.

There were fewer heme pigment casts, and they appeared to be disintegrating, and mixed with mononuclear cells and polymorphonuclear leukocytes. The glomeruli in some areas contained erythrocytes, but in general showed partial constriction and possibly increased cellularity (Figure 5). The interstitium remained normal. (It is probable that the state of constriction of glomerular capillaries as re-

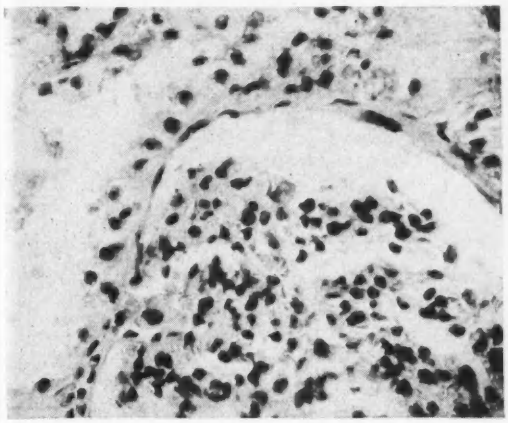

Figure 5 (Case 1).—Biopsy specimen (high power) at onset of diuresis, showing red blood cells in the glomerular capillaries, which are evidence of circulation through this structure.

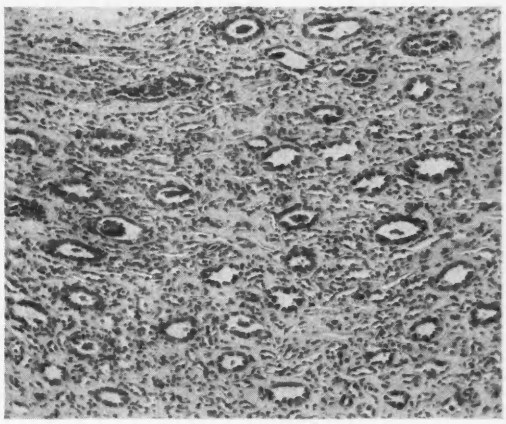

Figure 6 (Case 1).—Biopsy specimen (low power) at height of polyuria. Regenerating tubules are present.

vealed following the trauma of needle biopsy is not an accurate index of glomerular blood flow.)

Despite the initial appearance of destruction, careful examination of the specimen showed early regeneration and reconstitution of the tubules consistent with return of normal function. The presence of blood in the glomerular capillaries may have been evidence of circulation through these structures. Much of the pink-stained protein precipitate in the tubules had disappeared. This seemed to be evidence of passage of glomerular filtrate through the tubules.

Biopsy during the height of polyuric phase. The day preceding removal of a second specimen (from the right kidney) the patient eliminated 2,600 cc. of urine. This was the approximate peak of the polyuric phase.

Unfortunately this specimen was obtained from the renal medulla instead of cortex (Figure 6), and pelvic transitional epithelium was present (Figure 7). Cortical tissue was not present and comparison could not be made with the previous two biopsy specimens. Moderate amounts of eosinophilic intertubular ground substance were present. Collecting tubules were arranged in a regular fashion. Scattered neutrophilic polymorphonuclear cells appeared around the tubules along with an occasional eosinophil. Histologic preservation of tubular epithelium was excellent. Many tubules contained hemoglobin-colored casts mixed with nuclear and cellular debris and pyknotic polymorphonuclear leukocytes (Figure 8). In several areas the lining epithelium about these casts and elsewhere was irregular, serrated and partially desquamated. For the most part, however, the tubules were lined by a healthy appearing, single celled layer of cuboidal epithelium. There was little evidence of previous or present

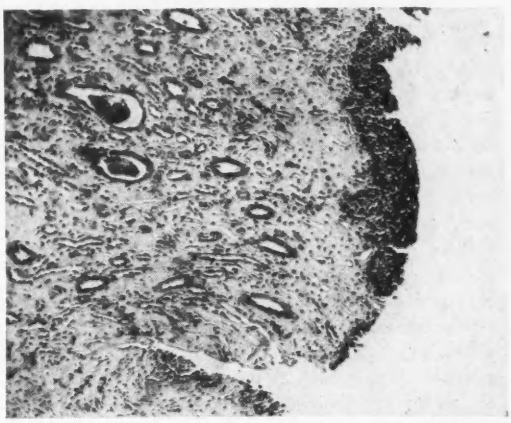

Figure 7 (Case 1).—Biopsy (low power) at height of polyuria. Transitional epithelium of renal pelvis included in biopsy; no evidence of urinary extravasation subsequently. Two tubules still contain casts.

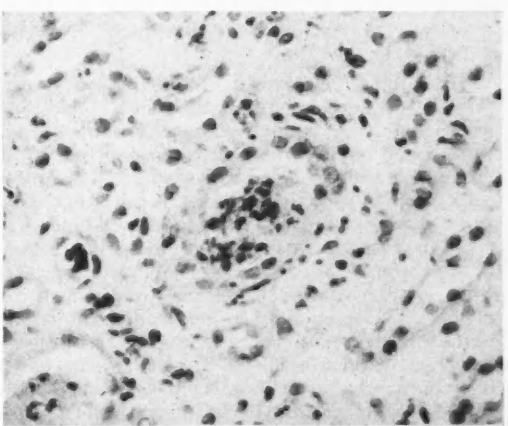

Figure 8 (Case 1).—Biopsy (high power) at height of polyuria. A disintegrating cast in a tubule.

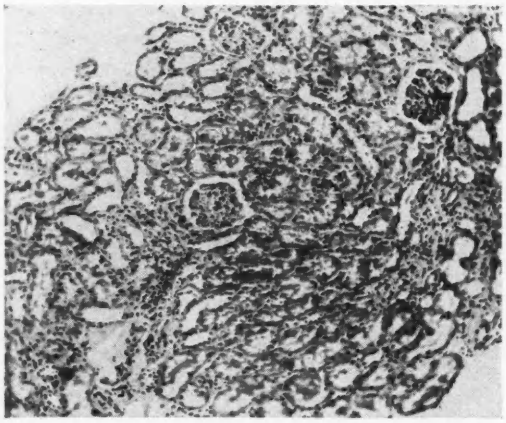

Figure 9 (Case 2).—Biopsy (low power) during diuresis in a patient with toxic nephrosis due to ingestion of carbon tetrachloride. Note patchy necrosis of some of the tubules. Clinical course and pathologic picture much more benign than Case 1.

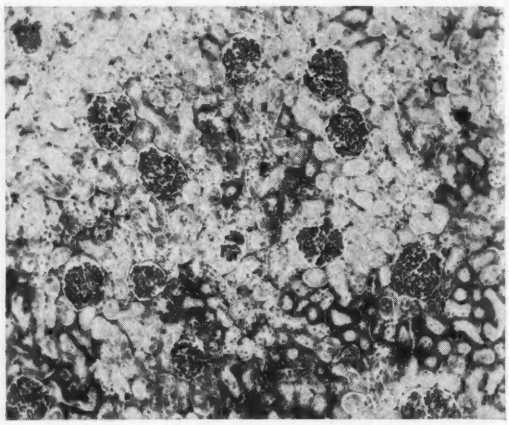

Figure 10.—Section of kidney of rat with experimental carbon tetrachloride poisoning (low power). Note the patchy tubular necrosis and normal glomeruli. (Courtesy of Dr. R. Jennings.)

injury to the epithelium of this portion of the nephron.

Oliver convincingly demonstrated anatomic reasons for profuse diuresis and failure to reabsorb essential electrolytes in the polyuric phase. By enzymatic stains of kidney sections from experimental animals, he showed an absence of mitochondrial rodlets in regenerating tubular epithelium. Mitochondria are thought to be concerned with the active transport (probably by phosphorylation) of essential substances. Thus the excessive loss of sodium, potassium, chloride, and perhaps water during the polyuric phase can all be explained by failure of the newly reconstructed tubular epithelium to adequately reabsorb these elements from the glomerular filtrate. Unfortunately, no conclusions can be drawn from this third biopsy specimen, as it only included renal medulla.

CASE 2. *Study of histologic features of the kidney during toxic nephrosis.*

The Urologic Department was requested to perform needle biopsy of the kidney in a 62-year-old white man who became oliguric two days after drinking 60 cc. of carbon tetrachloride while under the influence of alcohol. The period of oliguria was short, and by the fifth day the patient was excreting 1,500 cc. of urine. Needle biopsy of the kidney was performed on the twelfth day of illness. Upon study of tissue from the right kidney it was noted that some of the glomeruli showed fusion between glomerular loops and focal pink hyaline thickening of the basement membranes (Figure 9). In an occasional glomerulus that was involved, there was a distinct increase in the number of endothelial cells in the loop involved. The efferent arterioles occasionally contained proteinaceous pink precipitate. Many of the distal and proximal convoluted tubules contained desquamated granular debris or pink hemogenous vacuolated protein precipitate. An occasional tubule showed some evidence of epithelial degenerative change, although most seemed fairly intact. Several clusters of convoluted tubules were dilated, and the epithelial lining thereof thinned. Minute atrophic scars were seen occasionally in the cortex. In such foci, atrophic tubules were observed. In some distal convoluted tubules and proximal portions of the collecting ducts there were luminal, bile-stained granular casts. Occasional epithelial cells in those tubules contained cytoplasmic golden yellow-green, granular material.

The kidney damage appeared less severe in this patient than it was in the patient in Case 1, and in no areas were the nuclei of the tubular epithelium absent. Moreover the extreme degree of tubular dilation noted in the first patient (which, according to Oliver, is indicative of obstruction lower down in the nephron) was not present. The bile-stained material was due to the coexisting hepatic damage.

Also, the clinical course was more benign than that of the first patient. This may indicate that needle biopsy of the kidney in acute renal failure has value as a prognostic aid.

No ill effects attended renal biopsy and the patient had rapid recovery.

Experimental toxic nephrosis. Through the courtesy of Dr. R. Jennings of Chicago, we had an opportunity to study his sections of the kidneys of rats poisoned by carbon tetrachloride. While this lesion is not identical with the human lesion in toxic nephrosis, many points of similarity are present. Widespread evidence of proximal tubular necrosis was seen, with relatively intact distal tubules and glomeruli. The blood vessels and interstitium were not noteworthy (Figure 10). After nephrotoxic chemical poisoning, one would expect diffuse widespread lesions. Oliver's theory of generalized tub-

ular toxicity plus focal ischemia seems an adequate explanation of the observed focal lesions. He believed that only a vascular component can explain scattered lesions in the kidney after systemic administration of a nephrotoxic agent.

The similarity of this experimental lesion to the microscopic findings in the two cases reported herein seems to confirm nephrotoxic agents as the cause of renal failure.

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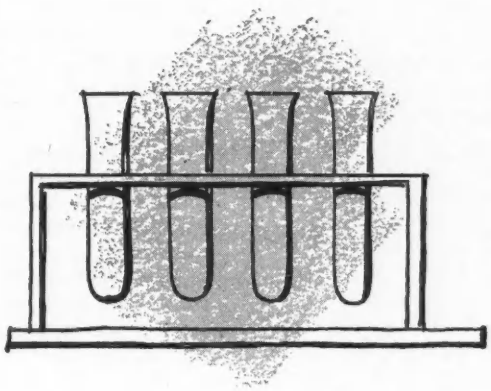

Cancer of the Liver

Needle Biopsy as a Means of Detection

JOHN W. FINDLEY, JR., M.D., and
BRADLEY C. BROWNSON, M.D., San Mateo

• *Needle biopsy of the liver was done in 25 patients suspected of having hepatic cancer. The results of biopsy were "positive" in 19 cases, and cancer later was proven to be absent in the livers of four of the other six. Multiple biopsy increased the incidence of positive findings. The procedure obviously provides no false-positive tests. It is recommended for the detection of hepatic neoplasm in patients who would otherwise require surgical exploration for diagnosis.*

NEEDLE BIOPSY is now established as a practical way to discover cancer in the liver. However, as yet too few physicians realize how frequently the suspected cancer is found and how easily the biopsy is done. The procedure entails a risk which is not negligible but becomes minimal when certain precautions are followed routinely.[2, 6]

In 111 patients with proven secondary or primary hepatic neoplasms, Ward, Schiff, Young and Gall[10] were able to demonstrate tumors in 82 by needle biopsy. Working in a cancer hospital, Bowden and Kravitz[1] found neoplastic liver disease in 42 of 76 patients with suspected hepatic metastasis. Sixteen of the remainder were later shown to be free of liver involvement, and in 18 cases the result of biopsy was falsely negative. Multiple biopsies in the same patient were employed rarely.

METHODS

In the series here reported, 36 liver biopsies were done in 25 patients, 10 men and 15 women, with clinically suspected but previously unproven hepatic neoplasms. The material was obtained with a Vim-Silverman needle. The age range of the patients was 39 to 82 years and the median age was 64. Twenty-two of the subjects had enlarged livers—in 16 cases they were arbitrarily considered "markedly" enlarged. In ten cases the liver was nodular. Three patients were icteric. The site of biopsy was subcostal in 19 cases, intercostal in three and both in three. One specimen was removed from each of 14 patients; two specimens from each of 11. No complications were encountered. Primary tumor sites are listed in Table 1.

RESULTS

Liver biopsy revealed neoplasm in 19 of the 25 cases in which there was clinical suspicion of cancer. Absence of hepatic involvement was proved in four of the remaining six patients, by laparotomy in two instances, by postmortem examination in one and by the subsequent course in one. In one patient, widespread melanosarcoma of the liver, primary in an eye, was not discovered by single biopsy. It is probable that a second biopsy would have been positive in that patient. In a patient with Hodgkin's disease without hepatomegaly, a single needle biopsy did not reveal the neoplasm. However, at autopsy, seven months later, the only evidence of liver involvement was one subcapsular metastatic lesion 3 mm. in diameter. In the other two patients without evidence of hepatomegaly on physical examination the liver was spared. Biopsies were positive in all of those with nodular livers and in two of the three icteric patients; the other patient with jaundice had a bile duct carcinoma that did not involve the liver. There were positive biopsy findings in all of the 11 patients in whom two biopsies were taken, but both were positive in only eight. In two of the three cases in which one specimen was intercostal and one subcostal, only one of the specimens was positive for carcinoma.

TABLE 1.—*Site of primary carcinoma in patients with liver involvement*

Colon	3
Liver	2
Stomach	2
Bile duct	1
Breast	1
Eye	1
Uterus	1
Hodgkin's	1
Undetermined	9
Total	21

Submitted June 6, 1955.
From the San Mateo Clinic.

DISCUSSION

Antemortem detection of hepatic metastasis by means of liver function tests is usually not reliable, although results may be suggestive.[7] Interest in ways of establishing the presence of tumor involving the liver has increased since the recent demonstration of a method using radioactive iodinated human serum albumin.[3, 8, 9, 11] In that method, gamma radiation emitted by hepatic tumor foci is measured with a scintillation counter. Although this method probably provides fewer false-negative results than liver biopsy, ascites, acute hepatitis and other conditions may be associated with false-positive findings. If resection of a primary tumor is to be foregone on the basis of evidence of a metastatic lesion in the liver, tests for the latter must provide no false-positive results. Although liver biopsy sometimes gives false-negative results, there can be no false-positive findings by this method.

The high incidence of positive liver biopsies is rather surprising. The usual concept of hepatic metastasis occurring only in widely separated nodules is not substantiated by the microscopic biopsy findings. Hoffbauer stated: "In the presence of metastatic carcinoma in the liver the biopsy, performed at the bedside, is of value only if the needle happens to strike a metastatic nodule."[4] This is of course true, but it has been shown that the needle does happen to strike metastatic foci with remarkable frequency. The incidence of positive results is higher if specimens are taken from more than one area. In the presence of hepatomegaly it is now the authors' practice to obtain two specimens when searching for malignant cells.

A question that arises is whether all patients with primary cancer of a kind that is known frequently to metastasize early to the liver, such as carcinoma involving the gastrointestinal tract, should routinely have preoperative liver biopsy. Unless a palliative surgical procedure is indicated, a positive specimen would preclude the need for laparotomy, with an attendant saving in discomfort, expense, hospital days and surgeons' time. Certainly in such situations liver biopsy appears indicated if the liver is enlarged and nodular. In the absence of enlargement, the indications are not yet clear. In this connection it is noteworthy that in a recent autopsy series, of 175 cases with hepatic neoplastic metastases, 36 per cent were not suspected of having liver involvement on the basis of antemortem abdominal palpation.[5]

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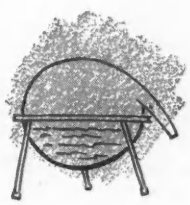

Extensive Burns in Children

Treatment of the Early Phase

EDWARD H. SENZ, M.D., San Francisco

• *The water, electrolyte and colloid losses and shifts which occur after a severe burning are complex, and variable from patient to patient. No available clinical method allows for accurate prediction of the volume and composition of the fluid losses. The method here described assumes that parenteral therapy which produces a normal urinary output and maintains the child free of signs and symptoms of shock is good therapy and at least approximates his needs. On the whole this assumption has been verified by clinical experience.*

Once again it is necessary to stress the importance of treatment that anticipates needs rather than treatment which comes after unmet needs are obvious.

Methods used to assess and treat burned adults need modification before application to infants and children.

A SEVERE BURN inevitably produces remarkable disturbances in the homeostatic mechanisms necessary to life. Rapid and inordinate losses and shifts of water, electrolytes and colloid material occur; and there may be considerable destruction of erythrocytes when the burn is deep.[5] These pathologic processes lead to shock and hypoxia and to all of the physiological aberrations secondary to shock and hypoxia. It is of utmost importance to recognize that the magnitude of the pathologic changes cannot be altered by mechanical or pharmacological means.[2, 6]

Although there is a difference in tolerance by different persons to burns of any given depth and extent, infants and children in general develop shock from burns of a depth and extent that ordinarily do not produce pronounced systemic effects in young, otherwise healthy, adults. At any time during childhood, a burn of over 12 per cent[7] of the body surface is likely to lead to shock, and in the first four to five years of life a burn of 8 per cent[1] or more of the body surface may be serious. In general, the younger the child the less well is a burn of any given depth and extent tolerated. Unfortunately, the general condition of the child shortly after the burning is likely to be misleading and overly reassuring. This apparent well-being may cause omission of measures needed to prevent or modify subsequent shock which then comes as a surprise to persons unfamiliar with the care of burned children. The later therapy is started, the less are the chances for success, for by the time shock has occurred, the losses of fluid, electrolyte and colloid already are incredibly large. Therefore it is necessary to anticipate the child's needs, since it may not be possible to catch up once shock has occurred.

All currently used methods of supportive therapy for burned patients require estimation of the extent and depth of the burn. This is not so simple as it might seem, particularly in the early phase of the burn, and serious errors of overestimation and underestimation occur. Judgment of the depth of a burn is more difficult than judgment of its extent, and the usual tendency is to underestimate depth. Although it is not the purpose of this paper to discuss local treatment of burns, it should be pointed out that with "open" or "exposure" treatment[7] the depth and extent of the burn can be reevaluated continuously and treatment changed accordingly; whereas, when bandages are used, treatment has to be based on the original and often faulty estimations. The "rules of nines"[7] which divide the body into areas representing 9 per cent of the surface or multiples thereof are most useful for determining roughly the extent of burns in adults. In using this method for children, however, serious errors arise, since a child has much more of his skin on the head and much less on the legs than does an adult. Therefore, the author prefers the data shown in Chart 1 as a basis for estimation of the extent of burns in children.[4]

The plan of treatment to be described is in no sense original, being based on the suggestions of Cope and Moore,[2] and having been used extensively in children by Alway.[1] The plan is simple and is based on clinical observations rather than on laboratory procedures that often are not available.

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Presented before the Section on Pediatrics at the 84th Annual Meeting of the California Medical Association, San Francisco, May 1-4, 1955.

CHART 1.—Surface area calculation (modified from Lund and Browder[4])

At all ages:

Neck	2 %	One foot	3.5%
One arm	4 %	Trunk	26 %
One forearm	3 %	Genitals	1 %
One hand	2.5%	One buttock	2.5%

Areas that change with age:

	At birth Pct.	At 1 yr. Pct.	At 5 yr. Pct.	At 10 yr. Pct.	At 15 yr. Pct.	Adult Pct.
Head	19	17	13	11	9	7
Thigh	5.5	6.5	8	8.5	9	9.5
Leg	5	5	5.5	6	6.5	7

CHART 2.—Plan for parenteral therapy

	Per Cent of Total Given in Each 12-Hour Period: 1st	2nd	3rd	4th
(a) *Loss to extracellular space (edema):* For maximal burn allow amount of fluid equal to: Under 3 years of age, 15 per cent of body weight; over 3 years, 10 per cent of body weight. For less than maximal burn allow amount of fluid equal to: Under 3 years of age, 8 per cent of body weight; over 3 years, 5 per cent of body weight. Whole blood, plasma, serum albumin, Ringer's lactate (no dextrose in water)	50	25	12.5	12.5
(b) *Surface loss:* Allow 50 ml. per 1 per cent burn/48 hours. Plasma, serum albumin, Ringer's lactate (no dextrose in water)	25	25	25	25
(c) *Maintenance:* 24-hour requirement ×2. 30 per cent Ringer's lactate, 70 per cent of 5 per cent dextrose in water	25	25	25	25

GENERAL TREATMENT

A catheter for intravenous infusion should be inserted at the earliest possible moment and before local treatment is started. A "cut down" and the secure placement of the largest catheter that the vein will hold saves costly later delays.

Since the plan of treatment is based largely on observations of urinary output, an indwelling catheter in the urinary bladder is essential so that output can be measured precisely. Without an indwelling catheter, retention or incontinence or both will destroy the most useful guide to the modification of treatment.

Placing the patient in an oxygen tent not only supplies additional oxygen to help combat hypoxia, but some patients seem to be more comfortable in a cooled tent, especially during hot weather.

Sedative and analgesic drugs usually are not needed in large quantities, and their depressing effects are undesirable. Surprisingly, most burned children seem little distressed, once handling has ceased. Restlessness and the appearance of discomfort often are signs of impending or actual shock and hypoxia. These conditions respond to antishock measures and not to opiates. Routine orders for opiates should not be permitted, and no depressing drugs should be given unless the physician is satisfied that pain rather than shock is the cause of a child's symptoms.

Adequate tetanus prophylaxis must be given.

Antibiotics in full therapeutic doses should be started early. Although in extensive burns some infection of the burned area almost always occurs, antibiotics can protect the patient against septicemia and against beta-hemolytic streptococcal infections of the skin which may cause failure of skin grafting[3] and/or nephritis.

Nothing should be given by mouth in the first 24 hours after the burning. Oral feeding often leads to vomiting and diarrhea which complicate the fluid and electrolyte problems. Gastric dilatation with respiratory embarrassment has been noted in cases in which oral feedings were given in the first 24 hours. The thirst of which some patients complain in the early period is a sign of impending shock[7] and is not a reason for offering oral fluids but a reason for intensifying parenteral treatment.

The vital signs should be watched closely and a detailed record of output and the composition and volume of fluid intake must be made.

Plan for Parenteral Therapy

Although it is known that the shifts and losses of fluids, electrolytes and colloid are great, no simple clinical means of estimating these shifts and losses for individual patients are available. Hence no formula can be expected to anticipate accurately the needs of all patients. Nevertheless, the following fluid plan is useful in estimating needs in the first 48 hours after a severe burning. It is convenient to plan for four 12-hour periods.

Losses to the expanding extracellular space—edema—((*a*) in Chart 2) may cause this space to expand 50 per cent,[2] which would be equal to 15 per cent of body weight in the infant and small child or 10 per cent of body weight in an older child. Expansion of the extracellular space is most rapid in the first 12 hours and usually is complete in 24 to 48 hours, when edema reaches its height. For this reason, half of the fluid calculated for expansion of the extracellular space is given in the first 12 hours and one-fourth in the next 12 hours. In this respect "first 12 hours" or "first 24 hours" means first 12 hours or first 24 hours after the burning, not after the beginning of treatment. If many hours have elapsed between the burning and the beginning of treatment, every effort should be made to get in the fluids calculated for the first 12 or first 24 hours in the time remaining, and careful

watch should be kept for signs of cardiac failure. For calculating the total quantity of fluid for losses to the extracellular space, it should be considered that a burn of 30 per cent or over probably will cause maximal possible expansion of the extracellular space, and an amount of fluid equal to 10 to 15 per cent of the body weight should be allotted. If the burn is under 30 per cent, an amount of fluid equal to 5 to 8 per cent of body weight should be allotted. If the burn is partial thickness, this fluid can be given as 50 per cent plasma (or serum albumin solution to avoid the risk of hepatitis) and 50 per cent balanced electrolyte solution (Ringer's lactate). If there is known or presumed full thickness burn, whole blood should be given in the first 24 hours in amounts equal to one-third to one-half the fluid calculated for extracellular space expansion. The hematocrit is not a very useful guide in gauging the need for blood. The need for whole blood in deep burns cannot be overemphasized, for regardless of what the hematocrit shows, the total erythrocyte mass is decreased[5] and the patient's oxygen-carrying capacity is diminished. A burned child will tolerate some extra blood better than a decreased erythrocyte mass.

Losses from the surface begin immediately and continue at a more or less even rate ((*b*) in Chart 2). For this loss, 50 ml. of fluid for each 1 per cent of area burned should be allowed in the first 48 hours; and one-fourth of the total can be given in each 12-hour period. These fluids should be given as 50 per cent colloid and 50 per cent balanced electrolyte solution. Since the relationship between per cent of burn and fluid requirement is not strictly parallel, the surface loss of fluid from burns of over 30 per cent should be calculated as though only 30 per cent had been burned.

Maintenance fluids ((*c*) in Chart 2) for 48 hours are calculated in the usual fashion, utilizing age or weight or surface area. Maintenance fluids are the fluids the child would need if he were not sick at all. These can be given as a solution containing 30 per cent Ringer's lactate and 70 per cent of 5 per cent dextrose in water with liberal amounts of vitamins B complex and C added. The maintenance fluids should be given at an even rate of one-fourth of the total in each 12-hour period. Chart 3 gives a convenient method of calculation of total maintenance fluid needs.

After the amounts of fluid for each need are calculated, they are totaled and in so far as the various fluids are compatible with each other, they are given simultaneously; or, if incompatible, they are given alternately, with blood and colloid solutions having priority.

Normal saline solution should not be used for any part of the treatment, since its high chloride content will intensify the metabolic acidosis that is likely to be present. In no circumstances can dextrose in water be considered as satisfactory replacement for surface losses or expansion of extracellular fluid.

CHART 3.—*Maintenance fluids per 24 hours for children (double quantities for 48 hours)*

Using Body Weight:
Allow 100 ml./kilo./24 hr. at 1 year.
Subtract 10 ml./kilo./24 hr. for each 3 years of additional age (e.g., 90 ml./kilo./24 hr. for 4 years, 80 ml./kilo./24 hr. for 7 years, etc.).

Using Age:
Allow 1,000 ml. total/24 hr. at 1 year.
Add 100 ml./year/24 hr. for each additional year (e.g., 1,100 ml. at 2 years; 1,200 ml. at 3 years, etc.).

Modification of Treatment in Accordance with Urinary Output

Once treatment has started, changes are made in accordance with urinary volume. A urine volume of 30 ml. an hour indicates that fluid replacement is satisfactory. If urinary volume rises sharply above 50 ml. an hour, the rate of fluid administration must be slowed. Urinary volume below 30 ml. an hour calls for a more rapid rate of fluid administration, since oliguria in the first 48 hours after burning almost always is due to underestimation of fluid needs. If there is a question as to whether oliguria is due to inadequate fluid administration or to a renal lesion, the infusion of 15 to 20 ml. of 5 per cent dextrose in water per kilogram of body weight in 45 to 60 minutes will produce a rapid increase in urinary output if no renal lesion exists, and demonstrate that the oliguria is due to inadequate therapy. It must be pointed out, however, that if treatment has not been started until after shock has occurred, urine output may not increase for many hours after the beginning of treatment. In this situation, treatment should be pushed, since the patient will die if fluid is withheld in the faulty belief that renal insufficiency exists. The basic plan is flexible and is easily stretched out or speeded up.

Edema

Rapid development of edema in the burned area and in the unburned area is to be expected if the patient survives. It is not due to overtreatment but is a part of the pathologic process initiated by the burning. The infused fluids do not cause the edema, but rather give the patient adequate materials to lose and yet maintain blood volume.

Courses After 48 Hours

Beginning at 48 hours, either of two courses may be noted: (1) if the burn is mostly partial thickness,

dramatic diuresis often occurs, with rapid disappearance of edema. If this occurs, the patient's fluid intake must be curtailed, but not the electrolyte intake, for electrolytes may be washed out in large quantity. Usually by this time it is possible for the child to meet his needs by oral feeding. (2) If the burn is largely full thickness, loss of edema is more gradual and there may be continuing need for high fluid intake if urine volume is to be maintained.

There are numerous exceptions and only close observation will determine the child's needs. It is at this point that the determination of serum electrolytes may be of great value in plotting further treatment.

Potassium Administration

Extra potassium is not needed in the first 48 hours, since it is being released in large quantities from damaged tissues. In fact, hyperkalemia may exist early, and potassium administration in the early period should be limited to that which is unavoidable if blood and plasma are to be used. After 48 hours, however, potassium excretion becomes great and hypokalemia will develop predictably unless extra potassium is given. When urine output is satisfactory, children may receive safely 3 milliequivalents of potassium per kilogram of body weight per 24 hours by vein and two to three times this amount by mouth.

EXPERIENCE

During a recent period of 34 months, 44 burned children were hospitalized on the Stanford Pediatric Service at the San Francisco Hospital. The burns have ranged in extent from 2 per cent to 70 per cent of the body surface. About one-third of the total number of children required supportive treatment of the type described. No deaths occurred. All of the children have been discharged from the hospital, although naturally some still are receiving plastic and rehabilitative therapy.

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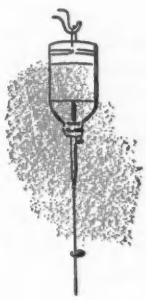

Injuries of the Fingers and Hands

A Review of Cases from the Standpoint of Compensation

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• Review of records in cases of injury to the hand that come before the California Industrial Accident Commission indicate that:

1. Primary closure at a suitable level in finger amputations is often preferable to plastic repair.

2. Complications incident to plastic repair in minor injuries frequently increase disability and cost to employer.

3. Tendon injury resulting from strain is a frequently overlooked cause of disability.

EACH YEAR more than 30,000 cases of injury come before the California Industrial Accident Commission staff—about 5 per cent of the more than 600,000 industrial injuries reported annually by California physicians. Those observed by the commission are the more serious disabling injuries, and those in which the question of compensation is contentious. In 8,000 of the 30,000 cases a year the injury is to an upper extremity—to the hand in the great majority.

In these cases commission staff physicians have had opportunity to see and to evaluate the final results of many types of treatment. While the commission has only limited facilities for statistical study, certain types of disability occur so consistently that the case histories come to assume a very definite pattern.

The present communication deals with a somewhat miscellaneous group of conditions in which the resulting disability seems unnecessarily high.

Finger Amputations

Amputation of a finger is relatively common and the final disability is often out of proportion to the loss.

The medical literature stresses conservatism in amputations of the fingers. It is repeatedly urged that finger length should not be sacrificed to facilitate primary closure and that length be preserved by the use of grafts and flaps.[3] The attitude is fostered that the resection of bone for the purpose of making a primary closure is almost a cardinal sin.

Apparently as a result of this teaching, many surgeons it seems are reluctant to resect enough bone to get an adequate amputation flap.

A workman's finger must be rugged enough to withstand considerable abuse. He must be able to maintain a firm grip on hard, heavy irregular objects. He must be able to thrust his hand into cramped inaccessible places without fear of bumping an exquisitely tender stump. In many of the cases that come before the commission the stumps are entirely unsuitable for this kind of use. In many cases the resection of a little more bone would have given the patient a rugged serviceable stump with a substantial reduction of permanent disability. Often in the cases reviewed, grafts have been used to avoid sacrificing length of finger. While grafts certainly preserve function in badly mutilated hands, the author feels that perhaps there is too great a tendency to use them for the sole purpose of saving a minimal length of bone.

Grafted fingers may look better but they are frequently unsuitable for laboring work. Loss of tactile sense is common and often the stump is very tender.

In order to cover exposed bone it is usually necessary to use a flap. This always results in more or less prolonged immobilization, and stiffness of the joints develops surprisingly often after this form of treatment, not only in the injured fingers but in the uninjured ones as well. It should be noted in this regard, therefore, that mere conservation of finger length is not necessarily consistent with conservation of function.

Plastic procedures certainly have their place and in many cases have made a hand usable. Before resorting to the use of a flap, however, the dangers and disadvantages should be carefully considered. It should not be forgotten that the resection of a minimal length of bone often allows for a good functional stump.

Fibrosis

Limitation of motion due to fibrosis is a frequent cause of disability. Although most physicians are aware of the danger of fibrosis in hand injuries, it is sometimes forgotten how quickly and insidiously it can develop.

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Immobilization in the presence of edema leads to fibrosis. The fibrin of serofibrinous exudates is deposited on gliding surfaces and around joints. The tissues are glued together.[9] Immobilization in hand cases is therefore always hazardous. Yet during the acute stage following injury the inflamed parts must have support and rest; otherwise increased tissue reaction with attendant edema may result. Prevention of fibrosis requires a very delicate balancing of rest and early mobilization. Elevation of the hand may help combat edema.

While the prevention of fibrosis in the injured parts may at times be impossible, much can be done to prevent stiffness from developing in the uninjured joints. Every uninjured joint of the extremity must be vigorously and continuously mobilized. It has been demonstrated many times that the patient will not do this on his own initiative. His natural instinct is to protect the extremity and he usually guards it well. Someone else must see to it that he puts every uninjured joint of the extremity through its complete range of motion every day. Time and again the Industrial Accident Commission reviews cases in which the surgeon apparently felt that his responsibility ended with instructions to the patient as to what to do.

Certain persons regardless of age are particularly prone to fibrosis. Who these persons are cannot be told in advance. By maintaining the hand in a position of function, however, it is possible to minimize the disability if it does occur. The position of function is with the wrist in 25 or 30 degrees of dorsiflexion and about 10 degrees of ulnar deviation, with the fingers partly flexed and with the thumb approximating the fingers. Then if stiffness occurs, the hand will be of some use even if motion is extremely limited.

Once fibrosis is present, long continued voluntary motion seems to be the treatment of choice. Gentle passive motion in the form of traction has a place. Vigorous passive motion probably does more harm than good.

Manipulation under anesthesia is practiced by many surgeons, who seem to feel that it is useful at times. Watson-Jones,[9] however, expressed the opinion that it is almost never indicated. Bunnell[1] said that forcing a stiff finger under anesthesia is dangerous and usually does harm. Results observed in cases before the Industrial Accident Commission have convinced the author of the uselessness of manipulation under anesthesia.

Loss of Grasping Power

Loss of grasping power is a ratable disability. Dynamometer readings are required in commission cases in which degree of disability is to be determined. There is no type of dynamometer that will measure exactly the amount of loss of grasping power. Dynamometer readings do not always reflect the actual loss resulting from the injury. If the examiner feels that they do not, he should say so and give an estimate, in percentage, of what he feels is the actual loss. The examiner should substantiate his opinion in every way possible.

The circumference of arm and forearm of both injured and uninjured side should always be reported. It has been noted that in persons doing active physical work, the circumference of the major arm and forearm is consistently greater than in the minor. Equal size usually means atrophy. It is usually arbitrarily assumed in cases before the commission that the grasping power in the major hand is 10 per cent greater than the minor.

Loss of grip can be caused by a number of things, among them amputation, limited motion of joints, tenosynovitis, nerve injury and pain. Evaluation of loss of grip is one of the most difficult problems of industrial medicine. It must be based on a number of factors, of which the loss shown by the dynamometer readings is only one. To be considered in every evaluation are the type of the original injury, the kind and duration of treatment, the complications, the time that has elapsed since injury, the objective findings, and pain. Loss of grip which is the result of pain is just as compensable in California as loss from amputation.

A reasonably accurate estimate of the loss which has resulted from amputation, from limitation of motion or from other cause requires a great deal of experience and judgment. Any preexisting loss of grip in the injured hand must be considered. As ratings are based on a comparison with the uninjured side, any preexisting loss on that side must also be considered.

Attempts to detect lack of cooperation by observing that the forearm muscles do not tighten or that the knuckles do not blanch, or any other such indication, are ordinarily useless. If there is no ability to grip, then there can be no tightening of the muscles and no blanching. The use of the blood pressure cuff in the taking of grip measurements is not acceptable, for by using different basal starting pressures almost any kind of a reading can be obtained.

It should be remembered that firm fixation of the wrist, and to some extent of the elbow, is necessary before a normal grip can be made. An example frequently seen is that of radial nerve injury. In that condition, while the flexor mechanism is undamaged, the paralysis of the extensors results in an almost total loss of grip.

The question of grip is one of the most difficult of the commission's problems. A subcommittee un-

der sponsorship of the California Medical Association has been set up to study the problem.

Tenosynovitis

Among the conditions frequently overlooked by industrial physicians are injuries to tendons and tendon sheaths brought on by stress or strain. The extensor tendons are most frequently involved. The extensors take part in any gripping action and, being weaker than the flexors, seem to be more subject to injury.

Tenosynovitis often occurs in locations in which a tendon passes through a sheath or tunnel under considerable strain. Increased pressure and friction bring on inflammatory changes and edema. In later stages a constrictive process may result. A common location is at the lower end of the radius where the abductor pollicis longus and the extensor pollicis brevis pass over the bone (De Quervain's disease). This condition occasionally results from roughness following fracture and it is frequently overlooked. Usually the condition is the result of strenuous gripping and pinching action.

The history is that of strenuous gripping or pinching from such activities as wringing out clothes, holding material up to a buffing wheel or difficult folding operations. A similar condition sometimes affects the ulnar side of the hand in typists who continually push the shift key with the little finger. The author once observed more than 50 cases at one time among a group of elderly women who had been recently put to work tying up bundles of electrical assemblies with heavy cord. The patients were not used to manual work of any kind and the job required very strenuous gripping all day long. The condition is much more frequent in women than in men. Diagnosis can usually be made by forced passive ulnar deviation during which the patient clutches his thumb within a tight fist[6] (Finkelstein test). The pain[7] in a positive test is quite severe and the patient's reaction is characteristic, and seldom simulated by a malingerer.

Tendon disorders may arise in locations where there are no sheaths. Excessive muscular fatigue causes reduction of glycogen to lactic acid. The lactic acid apparently trails down into the tendon with a lowering of pH and inflammatory changes with edema. Experiments have shown that actual necrosis of muscle fiber takes place. A lessening of the contractability of the muscle results. The entire process is a result of fatigue which may be due to repetitive motions without apparent strain. The patient is often unaware that his activities are causing the disorder. He often mistakenly blames some bump or blow. Crepitation is sometimes noted in conditions of this type, whereas, in the De Quervain type crepitation is unusual. The chief complaints are pain and loss of grip. The patient, usually a woman, may note that she "always drops things."

The treatment of these conditions is an immediate and complete cessation of the activity which brought it on. Rest by splinting cures many cases.[5] Operation is often necessary for disease of the constrictive types.[7] In the author's experience, heat and physiotherapy do more harm than good. The author has observed an extraordinary number of cases of the De Quervain type that occurred in persons carrying out enameling operations in which the operators strenuously gripped tongs all day long in the presence of extreme heat. The factor of heat is probably only coincidental.

If these conditions are recognized promptly, they are not much of a problem. Frequently they are not recognized and the patient is allowed to return to work too soon and the condition recurs. Frequently there is prolonged disability. Objective findings are minimal and the history, lacking any element of violence, often does not arouse suspicion. Frequently the patient is believed to be a malingerer.

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Epididymo-Orchitis

The Significance of the Condition in Industrial Surgery

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• Epididymo-orchitis, caused by direct trauma, is an uncommon condition, and the history and symptoms are quite different from those of the considerably more common so-called indirect epididymo-orchitis that is ascribed to trauma or strains, and which in symptomatology and clinical course is much like acute, nonspecific, pyogenic epididymitis. The author is convinced that the indirect trauma of muscular strain or minor injuries does not cause epididymitis; it is probable that slight trauma of that sort merely draws attention to an already existing inflammatory process that has made the scrotal area more sensitive than usual.

ACUTE, PAINFUL SWELLING of the testicles is a condition frequently observed by industrial surgeons. In the minds of patients and the physicians who examine them, the condition is so often associated with strain or minor trauma that the term *traumatic epididymo-orchitis* is frequently applied, which places it in the category of traumatic medicine. Yet the diagnosis of traumatic epididymo-orchitis is rarely justified when the facts are critically considered.

Infections of the lower genital tract (the prostate, seminal vesicles and posterior urethra), frequently spread to the epididymis, producing epididymitis in men of all ages and all walks of life. Inflammatory lesions of the epididymis, although most frequently due to nonspecific pyogenic organisms, may be due to specific infections, such as gonorrhea and tuberculosis, or may be metastatic in origin and follow bacteremia or septicemia. Frequently they develop after the use of retention catheters or instrumentation of the urethra.

In most instances, nonspecific epididymitis results from a progressive, nonsuppurative infection which has spread from an infection in the lower genital tract (the prostate, seminal vesicles, and posterior urethra), which may be apparent or without clinical manifestations, by way of the lymphatic channels of the vas deferens, or possibly by extension along the lumen of the vas deferens by direct continuity.

In certain circumstances infectious secretions from the lower tract as well as infected urine may ascend by way of the lumen of the vas deferens, when the walls are stiffened by inflammatory infiltration and the lumen is widened by cicatricial contraction and the ejaculatory duct is gaping. However, such a route is unlikely and urinary reflux through the lumen of the vas deferens is unusual. The normal ejaculatory duct prevents reflux of urine. The clinical picture of erroneously called indirect traumatic epididymo-orchitis is identical to that of nonspecific epididymitis, which is usually unilateral. The early initial symptoms of pain or discomfort in the inguinal region due to inflammation of the contiguous peritoneum, and swelling of the entire spermatic cord within the inguinal canal, are invariable. The initial inguinal pain is followed after a short interval by a more intense pain in the testis and epididymis. The skin of the scrotum becomes hot and red, but not ecchymotic, as the contents swell and become painfully tender to palpation or manipulation. Nausea and vomiting are of reflex nervous origin. Chills, fever, and prostration are due to absorption of toxic products coincident with the inflammation. Urinary symptoms, when present, are due to prostatitis, posterior urethritis, and cystitis.[3]

Herbut,[2] in describing the pathologic changes and complications of nonspecific epididymitis, stated that if the process is not extensive, it may completely resolve, but that generally it passes on to the subacute or chronic stage. The latter is represented by a disappearance of the congestion and the plastic exudate and by a complete replacement of dense, hyalinized, acellular, fibrous tissue. This completely replaces and destroys the epididymis and firmly unites this organ with the testis. Within the sclerotic tissue there may or may not be residual abscesses. When the infection is more severe, there is complete breakdown of tissue, with typical abscess formation. In recurrent lesions there is a mixture of the acute and subacute or chronic process. The testis, in cases of epididymitis, is generally normal. The most common accompanying lesions are urethritis, prostatitis and seminal vesiculitis. Local complications consist of incomplete resolution, hydrocele, abscess

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formation, relapse, atrophy of the testis, sexual neurosis, sterility (when disease is bilateral), and rarely thrombosis of the veins of the spermatic cord and gangrene of the testis.

The diagnosis of epididymitis is based upon the clinical history, symptoms, and physical findings. A urethral discharge harboring specific (gonococci) organisms, or the usual nonspecific pyogenic organisms, or the finding of pus, erythrocytes and pyogenic organisms in the centrifuged urine, associated with a prostatitis and seminal vesiculitis, is indicative of epididymitis not due to indirect trauma or strains.

Traumatic epididymo-orchitis implies direct (unilateral or bilateral) injury to the contents of the scrotum. Such a clinical diagnosis is tenable only when there is a history of a direct injury to the scrotum, followed immediately by a variable degree of shock, excruciating pain, swelling, and ecchymosis of the scrotum. Such an injury, with scrotal swelling, may go on to complete resolution or terminate in atrophy of the testicle.

Minor trauma to the scrotum and its contents is an every-day incident in males from birth until death. The concept that such trauma occurring at the place of employment on one occasion results in acute epididymitis, therefore would seem to be untenable. If the examining physician in such circumstances is diligent in his analysis of events immediately preceding such an episode and in his examination following the appearance of epididymitis, evidence of chronic prostatitis or infection of the posterior urethra preexisting the injury will usually be noted and the sequence explainable on the basis of the usual spread of such an infectious process from the lower genital tract. A more common sequence is that funiculitis and epididymitis have actually already occurred at the time of injury and that minor trauma of the scrotum such as might occur often and not be noticed is brought to the patient's attention because the scrotum has become hypersensitive.

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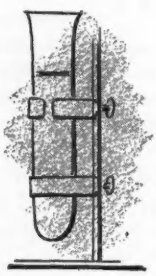

Tinea Capitis Due to Trichophyton Tonsurans

Incidence, Diagnosis and Epidemiology in the San Francisco Bay Region

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• *Eighty-five cases of tinea capitis due to T. tonsurans were observed in the San Francisco Bay area during the five years 1950-54.*

This disease, unlike the common microsporum infections, sometimes affects adults and adolescents.

Hairs infected with T. tonsurans do not fluoresce under the Wood's light. Diagnosis is a laboratory procedure in which the fungus is isolated from the hair.

There are three clinical varieties of the disease. The course is prolonged and treatment is unsatisfactory.

The disease apparently has spread from Mexico, through the Southwest and Southern California. Control is difficult.

SINCE MOST PHYSICIANS are familiar with the common microsporum ringworm of the scalp, the primary purpose in this presentation is to discuss tinea capitis caused by a species of fungus which has been observed recently in the San Francisco Bay area. This organism, Trichophyton tonsurans, has been found in tinea capitis in increasing numbers of cases throughout the United States, especially the Southwest, in the last few years. A large number of cases was reported from the Los Angeles area in 1952.[4,7]

Scalp infection by T. tonsurans does not always undergo spontaneous cure at puberty as do the microsporum infections. Consequently, this disease may be found in adolescents and adults. Trichophyton tonsurans-infected hairs, in contrast to microsporum-infected hairs, do not fluoresce under filtered ultraviolet (Wood's) light. Yet another characteristic of T. tonsurans infections is the great variation in clinical manifestations in different patients. While the most common clinical findings are scattered, irregular areas of alopecia, short broken hairs and pustular folliculitis, the clinical picture may vary from mild seborrhea-like scaling to severe kerion with scarring and permanent alopecia. For these reasons, along with its infectiousness, indolence and chronicity, tinea capitis due to T. tonsurans presents a most difficult diagnostic, therapeutic and epidemiologic problem.

INCIDENCE

One of the authors first made the diagnosis of T. tonsurans infection of the scalp in 1950, and since that time the disease has been found in 52 patients—42 from the Stanford Dermatology Clinic in San Francisco and ten from the north San Francisco Bay area who were seen in private practice. Thirty of the cases were diagnosed during 1954. During this same period 278 patients with microsporum infections of the scalp were observed. Thus, of a total of 330 cases during the past five years, 16 per cent were caused by T. tonsurans.

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Submitted April 5, 1955.

Table 1 shows the relative incidence of the various species of fungi in tinea capitis cases at the Stanford Dermatology Clinic and in private practice in the North Bay area. It appears that M. audouini causes most of the infections in clinic patients in San Francisco (69 per cent of 205 cases at Stanford) whereas in the North Bay region M. canis is more common. In a survey of dermatologists in the immediate areas surrounding most of San Francisco Bay, M. canis was reported most frequently as the cause of tinea capitis. Vallejo is probably a representative community in this respect, with 63 per cent of the cases due to M. canis. However, there are localities outside San Francisco, for example, Benicia, only 6 miles from Vallejo, where 83 per cent of the cases are due to M. audouini.

Thirty-three cases of T. tonsurans infections of the scalp in the five years covered by this study were reported in a survey the authors made of Bay Area dermatologists. These, with the 52 cases already mentioned, make a total of 85 cases of tinea capitis in this area from which T. tonsurans was isolated.

CLINICAL MANIFESTATIONS

Microsporum scalp infections occur almost exclusively in childhood and fall into two general clinical types: (1) Indolent, scaly, "gray patches,"

TABLE 1.—*The relative incidence of fungi responsible for tinea capitis in the San Francisco Bay Area (1950-1954)*

	M. audouini		M. canis		T. tonsurans	
San Francisco (Stanford Dermatology Clinic)	141	69%	22	11%	42	20%
North Bay Area	50	40%	65	52%	10	8%
Total	191	58%	87	26%	52	16%

Total cases 330.

TABLE 2.—*The characteristics of tinea capitis as seen in the San Francisco Bay Area*

	T. tonsurans	M. audouini	M. canis
Age	Adolescents and adults as well as children	Children	Children
Sex	More males	More males	More males
Spontaneous cure at puberty	No	Yes	Yes
Wood's light reaction	No characteristic fluorescence (infected hairs may appear dull gray)	Infected hairs green	Infected hairs green
Clinical appearance	Irregular bald patches interspersed with normal hairs. Scaling. Pustules and kerion common. Pruritic.	"Gray patches" of broken hairs. Inflammatory reaction infrequent.	"Gray patches." Inflammation. Pustules or kerion common.
Organism in hair	Large-spore endothrix	Small-spore ectothrix	Small-spore ectothrix
Source	Human	Human	Animal and human

single or multiple, with minimal inflammation. These are mostly due to the "human type" fungus, M. audouini, spread from child to child. (2) Inflammatory lesions, either pustular or with large boggy kerions associated with secondary bacterial infection. These are most commonly due to M. canis, which may be acquired from animals, particularly kittens. In both inflammatory and noninflammatory lesions infected hairs become lusterless and brittle and they break off or fall out. These hairs are more easily plucked than those not infected. Itching is not a common complaint. There may be enlargement of the posterior cervical nodes and pain with the pustular or kerion reaction. No attempt was made to determine how many of the tinea capitis patients observed by the authors had coexistent tinea corporis, but certainly a large percentage had lesions on the glabrous skin. Microsporum-infected hairs have a greenish fluorescence under a Wood's light.

Trichophyton tonsurans infections of the scalp may be clinically identical to those produced by the microsporum species. The hairs are not fluorescent. However, in 11 patients, infected hairs appeared dull gray under the Wood's light. The authors have been able to distinguish three clinical types of T. tonsurans infections which are suggestive but not diagnostic (Table 2):

Type I. SEBORRHEA-LIKE: Lesions with scaling or itching in irregular indolent patches resembling seborrhea (Figure 1). Often the patients are unaware of the disease because of the minimal symptoms. There is little loss of hair, and only by intensive searching, preferably with a hand lens, can a few broken hairs be found. There is little or no visible erythema. (With regard to this generalization, however, it should be noted that most of the patients we observed were Negroes.) The patches in the scalp are not well defined as in classical "ringworm," but are usually irregular or along the part in the hair, most often with moderately thick greasy scales. The "ringworm" pattern may be seen on the glabrous skin. Adolescent and adult patients who were observed showed this indolent form of the disease over a period of years.

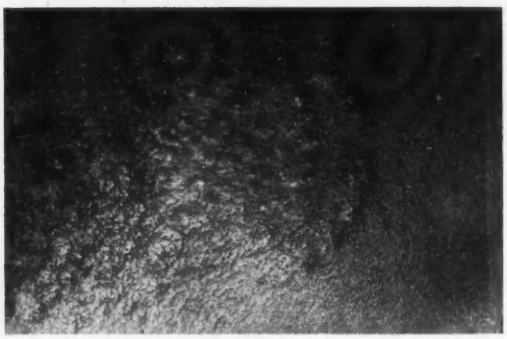

Figure 1.—Infected area at temporal hairline. Note the classical papulo-vesicular raised "ringworm" margin on the glabrous skin. This is absent above the hairline, suggesting a different immunologic response in the scalp.

Perhaps most significant is the paucity of signs and symptoms in this form of infection. Several of the children's parents brought the patients stating they could see lesions on the scalp, whereas grossly the authors could see little or nothing abnormal. The diagnosis was established only when the cultures were grown. It was often difficult to select a site from which to make the culture. Twelve (23 per cent) of the patients in the present series had this mild form of the infection. Five of the 12 were over 12 years of age.

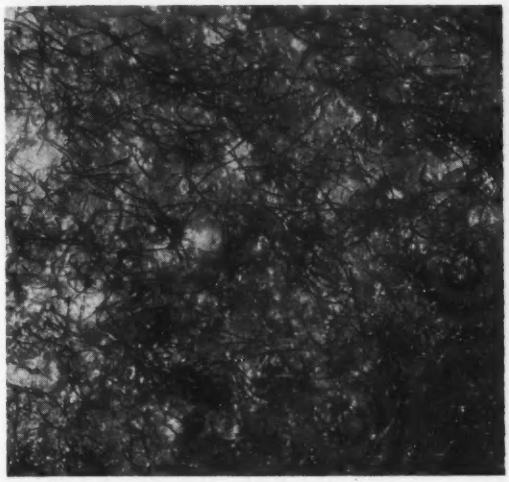

Figure 2.—Follicular pustules in Type II infection. No well-defined patch is found.

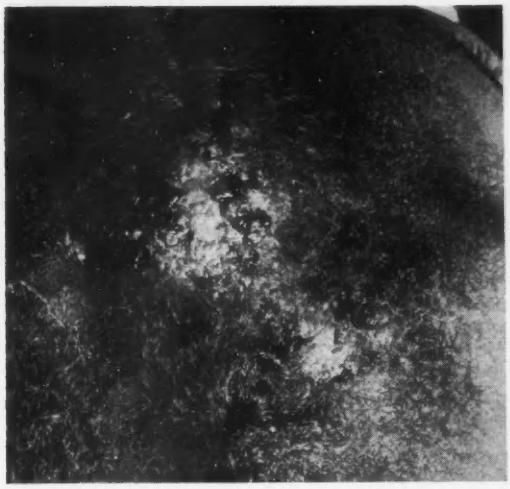

Figure 3.—Two kerions on the vertex of the scalp (Type III).

Occasionally a few "black dot hairs" were noted —brittle infected hairs broken off in the follicle at or just below the skin surface. These were sometimes covered by scales and were revealed only after shampoo or keratolytic medication removed the scales. In the present series only six patients had a great number of "black dot hairs."

Type II. PUSTULAR: Lesions consisting solely of follicular pustules which may appear sometimes in areas of erythema and alopecia (Figure 2). Some of these lesions were round or oval in shape, with accuminate pustules, while in other cases there were only a few pustules in lesions that otherwise resembled those of Type I. Occasionally long normal hairs appeared interspersed in areas of alopecia or in areas of short, broken infected hairs. Infected hairs are easily epilated manually whereas normal hairs are not. Twenty-four (46 per cent) of the patients in the series, all under 12 years of age, had this form of the disease.

Type III. KERION: Frank kerion formation associated with secondary bacterial invasion which shows severe tissue reactions and large boggy areas indistinguishable from the kerion of microsporum infections (Figure 3). Sixteen (30 per cent) of the patients in the series had lesions of this type. All except one were under 12 years of age. By comparison, kerion developed in 12 per cent of patients observed by us who had M. audouini and 18 per cent of those with M. canis infection.

Pipkin[6] called attention to the relatively high incidence of tinea capitis due to T. tonsurans in adults and adolescents. Three of the patients in the present series were adults (over 16), two were adolescents (12 to 15), and the remaining were children under 12. All the five patients over 12 years of age had the seborrhea-like form of the disease. The average age of the children was five years. The oldest patient was 62; the youngest, 2 years of age. There were 10 females, 42 males. Forty-six were Negroes and six Caucasians.

DIAGNOSIS

In none of the cases in the series could the diagnosis of T. tonsurans infection have been made without laboratory study. The clinical findings and lack of fluorescence could not be relied on alone, even by physicians familiar with the disease. Two procedures, readily applicable in office practice, are essential for diagnosis.

1. Direct examination of hairs or skin scrapings in potassium hydroxide. The hairs from suspected areas that are the easiest to pull out are the hairs most likely to be infected. Broken hair stubbles, if any are seen should be selected for examination. The hairs should be placed on a slide with a drop of 20 per cent potassium hydroxide and gently warmed (not boiled) or allowed to stand for about 30 minutes for "clearing." Examination should then be done under high dry magnification with subdued light.

Hairs infected with T. tonsurans are filled with broad strands of hyphae which readily break up into chains of large spores. These arthrospores vary from cuboidal to spherical in shape, are 4 to 5 microns in diameter, and are arranged linearly within the hair shaft. They are most numerous near the base of the shaft. This is infection of the large-spore endothrix type. In contrast, microsporum-infected hairs with a mosaic sheath of small spores

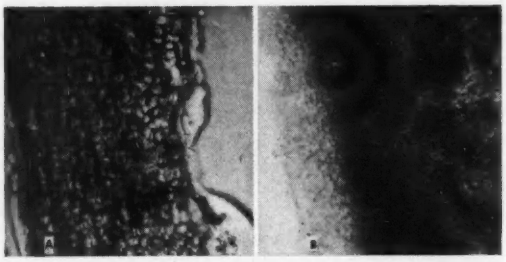

Figure 4.—*(a)* Large endothrix spores of Trichophyton tonsurans seen within the hair shaft. *(b)* Ectothrix spores of microsporum seen as a mosaic sheath on the surface of the hair shaft (×600).

surrounding the outside of the hair shaft have a small-spore ectothrix type of infection (Figure 4).

Scales removed from the scalp by scraping with a dull scalpel should be examined also in potassium hydroxide. These scales sometimes contain hyphal filaments. The hyphae appear identical to those in scrapings from any "ringworm" lesion, regardless of the species causing the infection.

2. Culture. Trichophyton tonsurans can be identified only by culture. It grows rapidly on Sabouraud's agar as white, yellow or tan velvety or powdery colonies. The reverse of the colony may be reddish-brown. The center of the colony may be crateriform. Microscopic mounts from the colony show numerous, large, clavate microconidia growing along the sides of the hyphae or on short lateral branches. The cell of the hyphae from which a microconidium develops generally remains unstained when mounted in lactophenol-cotton blue, whereas the microconidium takes a deep stain. Owing to the great variability in cultures, training in mycology is necessary for identification. Where facilities for proper identification are not readily available, cultures, hairs, or scrapings may be mailed to the State Department of Public Health Laboratory, the various medical school mycology laboratories, or to the Communicable Disease Center of the U. S. Public Health Service. Spores in dry hairs or scrapings remain viable for months or years at room temperature.

EPIDEMIOLOGY

Apparently T. tonsurans infections are transmitted directly or indirectly from human to human. No animal infections have been reported. Kligman and Constant[5] and others have reported family epidemics of T. tonsurans infections. The authors found 24 infected persons in nine families. Five members of one family, three adults, one adolescent and a nine-year-old boy, had Type I infections. All other familial infections were in preadolescent siblings.

Before World War II, M. canis was responsible for most cases of tinea capitis in the United States, mostly from animal sources, especially young cats and dogs, although human to human infections occurred frequently. Following the mass migrations of war workers and their families during World War II, epidemics of M. audouini appeared throughout the country. It is believed that these infections were carried from the large eastern cities, where they had been endemic for years.

Trichophyton tonsurans infection was known to be common in Mexico and Puerto Rico before World War II.[1, 3] There were scattered case reports from the East and Midwest until, in 1952, Pipkin reported a large series from Texas.[6] Since that time T. tonsurans infections have appeared in increasing numbers in the Southwest. Georg[2] expressed belief that these cases were owing to spread of infection from Mexico. Many cases have been reported in Southern California.[4, 7] Now the disease is appearing in Northern California in increasing numbers. Several of the patients in the present series had migrated from Texas and Oklahoma and many lived in housing areas near large government installations.

The Wood's light is of inestimable value in screening school children for microsporum scalp infections during epidemics. Veterinarians also have been alerted to its value in the diagnosis of microsporum infection in animals. Since Trichophyton-infected hairs are not characteristically fluorescent, it is readily seen that control measures are difficult to institute. Yet diagnosis and treatment is essential to prevent widespread epidemics. Nearly 25 per cent of the tinea capitis cases now seen in the Stanford Dermatology Clinic are due to this organism. In addition, eight infections limited to glabrous skin have been observed.

As was stated previously, M. audouini was the fungus most frequently isolated from cases observed in clinic practice seen in San Francisco and M. canis was the most frequently seen in private practice in areas surrounding the Bay. It is believed that T. tonsurans will be found more frequently if it is sought for and cultures are made of material from suspected cases. The technique for making cultures is simple and could be used for screening if school nurses and public health authorities were instructed. However, the cultures must be identified by a mycologist.

Enforced public health measures, including standardized methods of reporting, isolation and care of school children, and adequate instruction of school nurses and physicians should help control the spread of tinea capitis.

COURSE AND TREATMENT

The chief obstacle in treating and controlling T. tonsurans infections is the difficulty in obtaining adequate patient cooperation for follow-up. Patients with infection of Type I or Type II, with minimal, sometimes almost insignificant symptoms, neglect to return and often simply ignore the disease. Adequate patient education is difficult but important. For these reasons it was not possible to make adequate continuing observation of patients in the present series and hence obtain accurate statistics on the course of the disease. The criterion for cure is a negative culture. It is a certainty that the disease lasts for many months or several years. Two of the adult patients in the series had Type I lesions for at least eight years. It is most difficult to determine when a Type I infection is cured.

As in the microsporum infections, inflammation, either with pustules or kerion, is considered a good prognostic sign, as it indicates tissue response. In seven children with inflammatory (Type II or III) forms of the disease, spontaneous cure appeared to have occurred after 12 to 18 months. During most of this time topical fungicides were being applied, but it is questionable that this altered the course of the disease.

Because of poor patient cooperation plus the fact that Types II and III appear to undergo slow but definite spontaneous cure, the authors have not used x-ray depilation in cases of T. tonsurans scalp infections. It must be admitted that the treatment that has been used, consisting of topical applications of several of the many available fungicides, is not readily effective and that any cures obtained were most likely due to the patient's own immunologic mechanism. In the very chronic and indolent Type I infections, x-ray depilation probably is effective therapy.

Most of the authors' efforts have been directed toward controlling the spread of infection. The scalp should be covered at all times with a tight-fitting cotton stocking cap. The caps should be changed daily and the soiled caps boiled. The hair should be cut short (one-fourth inch or less) preferably with clippers, and it should be thoroughly shampooed at least twice weekly. Shampooing after clipping is important in order to wash away infected hairs. Hair clipped off should be burned. The head of the clipping instrument should be sterilized after using, preferably in phenol solution. Care should be taken with bedding, towels and headwear. Close contact with other persons must be avoided as much as possible. Fungicides in an ointment base applied twice daily to infected scalps probably suppress the spread of infection to others. Most important is the careful examination with culture of all members of the family and others in close contact with the infected person. In addition, patients should be cautioned against having haircuts in public barber shops. It has been our policy to allow infected children to attend school and to notify the school health authorities. Only in rare instances—when proper care and cooperation could not be obtained—has it been necessary to exclude infected children from school.

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CASE REPORTS

Pulmonary Alveolar Adenomatosis

Report of a Case

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PULMONARY ALVEOLAR ADENOMATOSIS has been regarded as a variant of alveolar cell (bronchiolar) carcinoma.[9] The tumors differ from bronchogenic carcinoma in their multicentric origin, in their relatively benign microscopic appearance and in that they rarely metastasize.[9] Clinically, the condition simulates chronic low-grade pulmonary infection with progressive involvement of the lungs and fatal termination.[2] On the x-ray film the lesions are either pneumonitic or nodular[8, 11] in appearance. Microscopically, hyperplasia of the alveolar epithelium and metaplasia into columnar mucus-producing glandular tissue[2] can be observed. Curiously, pulmonary adenomatosis resembles a viral respiratory disease of sheep that is called Jaagsiekte.[5, 6] The infrequency[14] of proven instances of this condition prompted the following case report.

REPORT OF A CASE

The patient was a 46-year-old Chinese-American farmer who resided in a suburb of Oakland, California. Except for yearly bouts of "flu" lasting for one to three days, he had always been well. During the second World War he was an electric arc welder in the shipyards for approximately four years. He said he did not use tobacco. The present illness began as a "chest cold" in April 1951 with a productive cough aggravated by exertion. An ounce of mucoid grayish white sputum was raised in 24 hours. The patient had pleuritic pain in the right side of the chest and wheezing on recumbency. There was no history of hemoptysis or of streaked sputum. At the time the patient was first examined in November 1951, the foregoing symptoms had persisted some seven months. In addition, the body weight had decreased 10 pounds and moderate dyspnea was present.

The patient was well developed and appeared to be in reasonably good health. Upon physical examination the only noteworthy abnormalities observed were of areas of consolidation over both lung bases. There was no enlargement of lymph nodes and no skin lesions. Results of skin tests for tuberculosis, coccidioidomycosis and histoplasmosis were negative.

Submitted May 27, 1955.

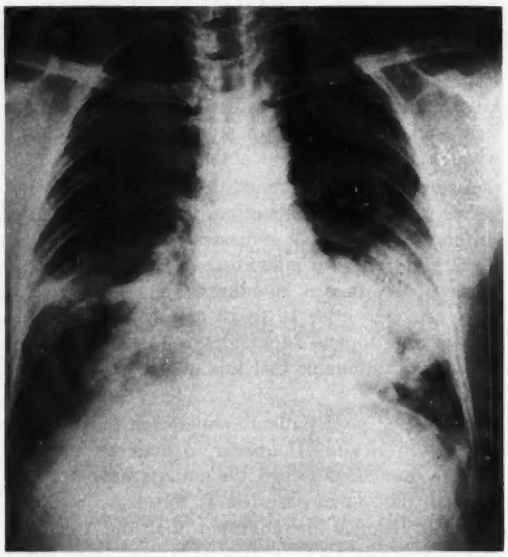

Figure 1.—X-ray film of chest December 3, 1951, showing bilateral diffuse pneumonic infiltration.

Roentgenograms of the chest showed a diffuse pneumonic type of bilateral pulmonary infiltration (Figure 1). Examinations of sputum and material from the stomach by concentration, smear and culture were negative for tuberculosis. The hemoglobin content of the blood was 15 gm. per 100 cc. and erythrocytes numbered 5,050,000 per cu. mm. Leukocytes numbered 11,900 per cu. mm.—81 per cent polymorphonuclear cells and 19 per cent lymphocytes. The sedimentation rate was 25 mm. in one hour (Westergren). Serum albumin was 3.7 gm. and globulin 3.1 gm. per 100 cc. Cholesterol content was 227 mg. per 100 cc. Blood chlorides were 420 mg. per 100 cc., or 73 milliequivalents.

On bronchoscopy no tumor or other abnormality was observed. The report on a Papanicolaou test was "Group 3, doubtful." Biopsy of a superior mediastinal lymph node was negative for disease. Exploratory thoracotomy was performed and the right middle lobe was removed.

Pathologist's Report. The entire middle lobe of the right lung was indurated, rubbery in consistency

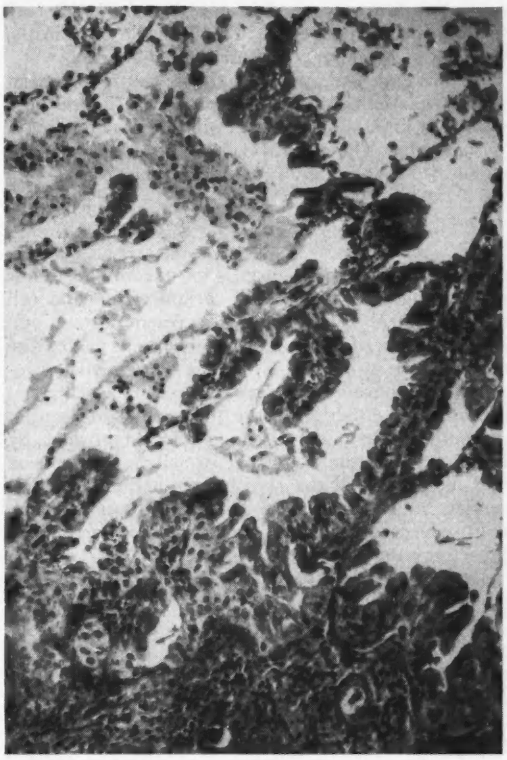

Figure 2.—Section from tumor mass removed at lobectomy. Note the adenomatous appearance of the lung. (× 100).

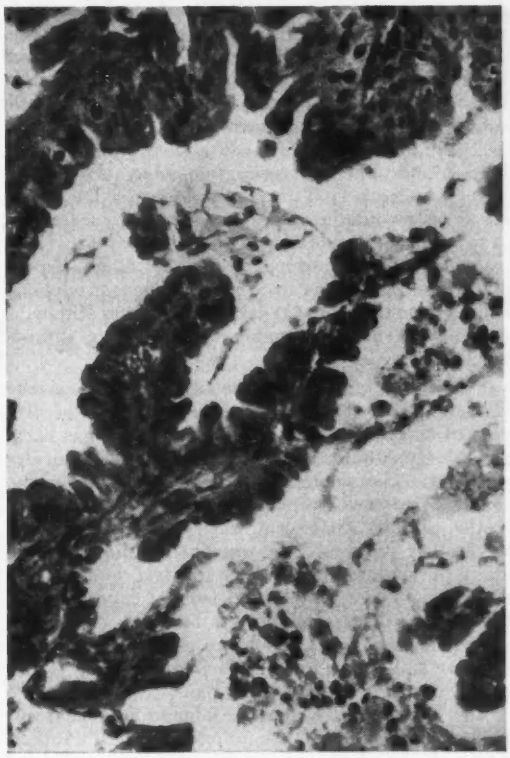

Figure 3.—Same section (× 430). The deeply staining tall columnar epithelium is striking.

and somewhat grayish in color. Sections (Figures 2 and 3) from various areas of the lobe were similar one to another in microscopic appearance. The lesion appeared to be diffuse. There was pronounced hyperplasia of deep-staining, high columnar epithelial cells lining the alveoli. The lining epithelium appeared to invaginate into papillary folds and adenomatous masses. There was an abundance of mucinous substance into the intra-alveolar spaces. The basement membrane was preserved. No mitotic figures were seen. The impression was pulmonary adenomatosis.

The patient was given 1,000 r of deep x-ray radiation in the depth of each lung. However, the condition of the patient deteriorated. Cough and dyspnea increased; the sputum became frothy, mucoid and abundant; weakness and loss of weight continued. Examinations of the blood showed progressive anemia. The disease process as observed roentgenographically increased in extent (Figure 4). The patient died a year after the onset of symptoms. Permission for autopsy was not granted.

DISCUSSION

The subject of pulmonary adenomatosis has been ably discussed by Wood and Pierson,[15] Delarue and Graham,[3] Paul and Ritchie,[11] Weir[14] and many

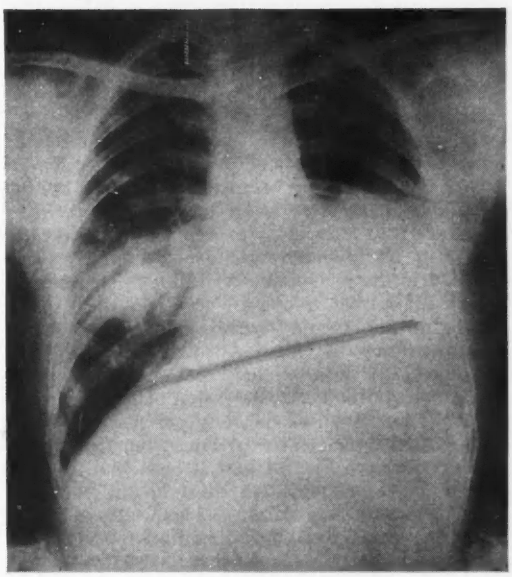

Figure 4.—X-ray film of chest taken after excision of the right middle lobe. Note increase in extent of tumor mass in the left lung.

others.[4, 12, 13] Weir reviewed 70 reported cases (of which he accepted 33) and then himself added reports of three cases. Bubis and Erwin[2] described the clinical picture accompanying this lesion as follows: There is a long, slowly progressive downhill course with persistent bronchopulmonary symptoms of low grade infection. No causative organisms have been found. The roentgenograms may show little change over a period of months or years. The lungs are grossly voluminous and heavy with raised moist gray patches of tumor. Microscopically, there is a transformation of normal alveolar lining by hyperplasia and metaplasia to a benign mucus-producing columnar epithelium. Ikeda[8] and Paul and Ritchie[11] described a multiple nodular form and a diffuse pneumonic type.

Most investigators have mentioned the remarkable similarity of pulmonary adenomatosis to a disease of sheep known as Jaagsiekte, which has been extensively studied by Dungal[5, 6] of Iceland. In sheep, the characteristic symptoms are slowly increasing dyspnea accompanied by excretion of a frothy, slightly opalescent watery mucus from the respiratory passages. The onset is insidious with a lapse of six months between contact and visible symptoms. There is no fever. Dungal expressed belief that the disease in sheep is probably caused by a pneumotropic virus which grows intracellularly in alveolar and bronchiolar epithelium. The sheep disease is not transmissible to humans.

The majority of writers on human adenomatosis have said they believed the lesion is neoplastic rather than infectious. However, the epithelial-like lining cells seen in this condition may be produced in the lung by a variety of other conditions,[1, 7] by foreign bodies, by tar and tar derivatives, by chronic passive congestion, by various kinds of viral and bacterial or chemical pneumonia and by x-ray irradiation. As a matter of fact, cells of this kind may be seen in the lining of the alveolar wall in so-called normal lungs.

Although the clinical manifestations resemble those of progressive fatal pulmonary infection, it is the replacement of normal lung tissue by the expanding tumor mass—simply, cancerous growth—which leads to anoxia, dyspnea and eventual death. This can be observed roentgenographically, particularly in the pneumonic form, in which massive, solidly infiltrative lesions are seen and where there can be no doubt how internal asphyxia occurs. In sharp contrast to the singularly pulmonary origin and involvement of this lesion is that of bronchogenic carcinoma. In the latter, the lesion originates in the bronchus and involves the lung parenchyma either by obstructive atelectasis, by contiguous growth or by infectious sequelae of partial bronchial obstruction. Finally, adenomatosis must be differentiated from metastatic lung carcinoma as well as chronic aspiration pneumonia including lipoid pneumonia.

Since adenomatosis is often confined to one lobe and metastasis is rare, it may be curable. Hence, prompt diagnosis is important. The lesion ought to be kept in mind at all times. Resection in unilateral cases will not only establish the diagnosis but may prolong or even save life. This was the case in the patients of Woods,[15] Graham[3] and Osserman.[10]

In the case here reported there was involvement of multiple lobes at the time of diagnosis and cure then was impossible. X-ray radiation was of no apparent value. The clinical course in the present case resembled that usually reported in other cases.

SUMMARY

Pulmonary adenomatosis is an uncommon tumor of the lung. It is probably a variant of alveolar cell carcinoma. Morphologically, it resembles an epizootic disease of sheep that is called Jaagsiekte. The clinical course is similar to that of persistent progressive low-grade pulmonary infection. Roentgenograms show either a diffuse pneumonic infiltration or multiple nodules. Diagnosis is established by tissue examination following excision. Hyperplasia and metaplasia of the alveolar epithelium into columnar, mucus-producing glandular tissue can be seen microscopically. If the disease is unilateral, lobectomy may prolong or save life.

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Coccidioidomycosis in Dogs

A Report of Three Cases

NORMAN E. LEVAN, *M.D.*, and
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IT IS THE PURPOSE of this paper to present three clinical cases of coccidioidomycosis in dogs, and to discuss briefly the potential epidemiologic significance of such infections in household animals.

CASE REPORT

CASE 1. On November 28, 1953, a previously healthy 18-month-old female Scotty was hospitalized with a three-day history of trembling, anorexia, fever and slight cough. The dog had been born and reared in Taft, California. There was no history of previous illness. Findings on admission were those of a respiratory tract infection, with temperature of 104° F. rectally (normal 101.5°). During the next few days despite therapy including anticanine distemper and hepatitis serum, dihydrostreptomycin and penicillin intramuscularly, and sulfonamides orally, cough increased and dyspnea was noted. No further antibiotics were administered. In the following two weeks on supportive treatment only, although intermittently febrile, the dog improved sufficiently to permit discharge from the hospital. Subsequently C. immitis was isolated on culture of sputum taken December 10, 1953.

Roentgenograms of the chest on December 15, 1953, showed an area of infiltration overlying the right diaphragm. No enlargement of nodes or bone lesions were noted. A follow-up roentgenogram taken February 28, 1954, showed complete clearing of the previously described infiltrate. On October 5, 1954, eleven months after the onset of illness, a complement fixation test for coccidioidomycosis showed a four plus reaction in dilutions through 1:24. In the meantime the dog had become and remained asymptomatic.

Comment: From this case it is apparent that the self-limiting "Valley Fever" syndrome, so common in humans in endemic areas, occurs also in dogs and can be diagnosed in a similar fashion. Thus a presumptive diagnosis of primary pulmonary coccidioidomycosis could be made in this case on (1) the appearance in a highly endemic area of an acute respiratory disease that did not respond to treatment ordinarily effective in bacterial infections, (2) roentgenographic findings consistent with the diagnosis, and (3) the concurrent appearance in a kennelmate (Case 2) of a similar syndrome which progressed to histopathologically proven disseminated coccidioidomycosis. Final proof rests on the finding of C. immitis in the sputum and the presence of highly specific serological antibodies in significant titer.

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Submitted March 25, 1955.

CASE 2. On October 23, 1953, a seven-month-old male Scotty (kennelmate of the dog in Case 1) was hospitalized with a history of listlessness and anorexia of a week's duration, and possibly anuria during the preceding day. A distressed appearance and a slightly elevated temperature were noted at the time of admittance. The leukocyte content of the blood was greater than normal.

Following catheterization at the time of admittance the dog was able to void normally. During the next four days he received intramuscular injections of penicillin, dihydrostreptomycin and vitamins. Nevertheless the temperature rose to 104° F., there was slight loss of weight, and ascites appeared. During the ensuing month there was continued deterioration in the dog's condition despite administration of aureomycin orally and sulfonamides intravenously, forced feedings during periods of anorexia, and the administration of oxygen. On postmortem examination lesions of disseminated coccidioidomycosis were found in the lungs, mediastinal lymph nodes, myocardium, kidneys and adrenals.

Comment: This case was observed clinically from its onset as an acute respiratory infection through its subsequent dissemination and fatal termination. In this instance the disease ran a rapidly fulminating course.

CASE 3. On February 27, 1954, a previously healthy year-old male boxer, born and reared in Bakersfield, California, was hospitalized because of a two-day illness characterized by dyspnea and anorexia. The symptoms noted at admission were those of a respiratory tract infection with temperature of 105° F. rectally, and moist râles throughout both lungs. During the next month, in addition to supportive care, the dog received successive courses of the following drugs: Sulfonamides intravenously and orally, penicillin and dihydrostreptomycin intramuscularly, and aureomycin, chloramphenicol and erythromycin orally. There was partial clearing of the lungs but the dog remained febrile.

In view of the failure of these therapeutic agents, ordinarily effective in canine pneumonia, the possibility of coccidioidomycosis was entertained as early as the first week of treatment. On April 1, 1954, serologic tests for C. immitis infection showed complement fixing antibodies in dilutions through 1:32 and precipitins in dilutions through 1:40. These tests were performed at the School of Public Health, University of California, under the supervision of Dr. C. E. Smith, who reported, "While the specimen was anti-complementary and thus adequate interpretation of the complement fixation test is not feasible, precipitins offer convincing proof that [the dog] had coccidioidal infection."

The animal was next seen September 28, 1954, when he was brought in for euthanasia. In the interim there had been progressive wasting, and gradual enlargement of hard, apparently painful swellings on the legs which had been first noted by the owner in May, 1954. Before the dog was killed a blood specimen was sent to Kern General Hospital.

It showed a four plus reaction to the complement fixation test for coccidioidal infection in dilutions through 1:16. A roentgenogram of the chest showed a small calcific density at the right base, increased markings paralleling the right cardiac border, and evidence of rib destruction which had repaired by massive formation of callus. Roentgenographic examination of the right hind extremity showed a calcific overgrowth involving the entire distal fourth of the tibial shaft.

On postmortem examination the essential findings were coccidioidal granulomas of the lungs, particularly the pleural surfaces, the bones and the liver.

Comment: In this case the dissemination was manifested as a much more chronic process than in Case 2. There was a slow downhill course with granulomatous metastastic lesions.

DISCUSSION

Naturally occurring coccidioidomycosis, as contrasted to cases resulting from laboratory inoculation, has previously been reported in many animals including wild rodents, cattle, sheep, monkey and gorilla, chinchilla, and six times in dogs. In each the diagnosis was made on necropsy; thus Cases 1 and 3 reported herein are the first in which the diagnosis was established in a living animal.

That three cases of canine coccidioidomycosis were recognized at one veterinary hospital in a brief period indicates that the paucity of reports is not a true reflection of the frequency of the disease in pets. Inasmuch as dogs may have a self-limiting pulmonary infection (Case 1) it is probable that numbers of such cases in particular are going unrecognized. The question arises whether household transmission of coccidioidomycosis is possible. (In this regard recent reports[1, 2] dealing with histoplasmosis in animals and man on the same farm are of interest.)

Rosenthal and co-workers[4, 5, 6] proved in several significant experiments that sputum from infected animals can constitute a hazard. They showed that spherule-containing exudates could produce pulmonary lesions when instilled endotracheally. Further, when spherule-containing sputum was exposed under a variety of conditions, viable forms, both vegetative and parasitic, could be found for long periods. In addition they found that normal animals when housed with guinea pigs having pulmonary coccidioidomycosis, developed the disease. A similar means of infection is implied in a case report[3] of coccidioidomycosis in a dog never outside of Canada, after exposure to a dog from California.

SUMMARY

Three cases of coccidioidomycosis in dogs are presented. They demonstrate that a parallelism exists between canine and human manifestations of the disease, including the self-limiting "Valley Fever" syndrome.

Roentgenograms were interpreted by J. W. Birsner, M.D., Radiologist, San Joaquin Hospital, Bakersfield.

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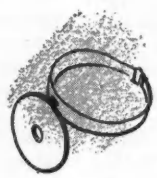


California MEDICINE

For information on preparation of manuscript, see advertising page 2

EDITORIAL

Professional Liability

ONE IN TWELVE doctors has a malpractice claim of some kind levied against him each year, according to analysts of the Northern California professional liability program. Some of these claims become lawsuits. Some of the suits are not successfully defended. The dollar size of judgments awarded by the courts increases enormously from year to year.

Trying to keep pace with the acceleration in claims, insurance companies have doubled and trebled premiums. But with premium increases always a year behind increases in the number and size of claims, the losses of the insurance companies have been staggering. They have progressively withdrawn from the field. Some insurance people hold the opinion that the day will come when physicians will be unable to get professional liability insurance at any price because there will be no insurance companies willing to underwrite the risk.

The medical profession, however, cannot withdraw from the problem of professional liability. Physicians will have to continue to live with it. Medical malpractice is therefore primarily a problem of medicine. It is not something the profession can leave to the insurance companies. It demands the interest and cooperation of every physician. It demands the study and effective, intelligent action of every medical organization.

For these reasons the California Medical Association accepted a responsibility for medical malpractice at the last meeting of its House of Delegates. The House created a Medical Review and Advisory Board to investigate and analyze all aspects of professional liability, to inform and advise the California profession, to make recommendations for effective action in this field and to cooperate with county societies and offer coordination of existing programs.

Ten physicians with a wealth of knowledge of professional liability matters in every part of the state have been named to the Board, with Joseph F. Sadusk, Jr., M.D., of Oakland, as its chairman and Wilbur Bailey, M.D., of Los Angeles, as its vice-chairman. The Board is well advised. It has retained the services of Joseph Linder as actuarial consultant. Mr. Linder guides the insurance aspects of the New York state malpractice insurance plan, the largest in the nation. Howard Hassard, C.M.A. legal counsel, whose law firm has nearly a half century of intimate experience in California malpractice, advises the Board on the legal aspects of the problem. Rollen Waterson, who originated the Northern California plan in Alameda County, serves the Board as executive secretary.

A section of CALIFORNIA MEDICINE devoted to the problems of professional liability makes its first appearance in this issue under the heading, "Memo from the Medical Review and Advisory Board." Dr. Sadusk's first contribution to the section, "What Price Medical Malpractice Insurance," will excite a great deal of interest, spirited comment and some controversy. He paints a dismal picture—perhaps too dismal. However, his experience and research in the field have been wide and thorough. We cannot ignore his dire predictions. Certainly any physician who has a sense of complacency in the matter of professional liability will have lost it before he has read many paragraphs of Dr. Sadusk's paper.

Certain of his conclusions deserve emphasis here:

1. The physician who selects his malpractice insurance coverage on the basis of price alone invites disaster. Factors of prime importance are stability and integrity of the insurance company, limitation of clauses in the contract detrimental to the physician, permanence for yearly renewal of coverage, and adequate reserves in the United States for paying claims or for attachment in the event of a disagreement at some future date between carrier and physician. Malpractice actions may be—and are—brought against physicians many years after the actual incident occurs. The physician, therefore, must be certain his company will still be available to defend him and, if necessary, to pay a claim.

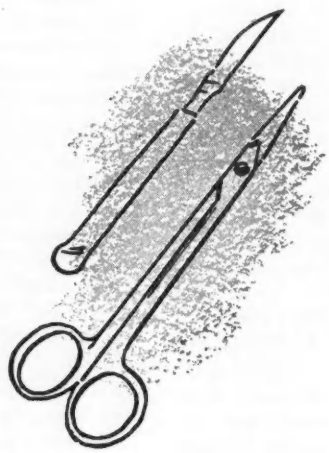
2. The low limit coverage of years ago is unrealistic today; physicians will do well to heed the author's advice, which is based upon observation of actual emotional and economic ruin of physicians who were inadequately protected.

3. The grouping of physicians for protection in this type of insurance is of benefit to all concerned. Only through a group plan can the physician be assured of the vital advantages of adequate investigation of claims, experienced defense, group negotiation with the carrier of contract and premiums, and other assets outlined in detail by Dr. Sadusk.

4. The problem is medicine's, and can be solved only by medicine. The most effective contribution to solution will be made when all physicians give full attention to the principles of "good faith, good records and common sense" as described in Dr. Sadusk's paper.

The new section on professional liability will appear periodically in future issues of CALIFORNIA MEDICINE. In addition to reports of the Medical Review and Advisory Board, specific cases will be described and informed medical, insurance and legal opinions on malpractice will be communicated to the profession.

The assignment given to the Medical Review and Advisory Board is as vital as it is big and difficult. Absolute success is impossible, but reduction of claims and premiums and adequate protection for the individual physician can be achieved through informed cooperation of physicians with their county society programs, and cooperation of county societies with C.M.A.'s Medical Review and Advisory Board. Reading Dr. Sadusk's interesting paper, on page 389, is the first step toward the informed cooperation necessary to solution of the growing malpractice program.



LETTERS to the Editor . . .

The Salk Vaccine

EDITOR'S NOTE: *The opinions expressed are those of the author, not of* CALIFORNIA MEDICINE. *For the California Medical Association's position on this subject, see* CALIFORNIA MEDICINE, *June 1955, page 465.*

THIS COMMUNICATION will endeavor to present some points unmentioned in that large fraction of writing so strongly dedicated to a misguided optimism. I am not a physician, public health employee, owner of stock in or employee of commercial laboratories, or in any way an affiliate of Mr. Basil O'Connor, of Dr. Jonas Salk, or of federal government laboratories. However, I have endeavored to present the subject of vaccines to medical and pharmacy students for over 30 academic years; I have worked in and visited plants of producing laboratories; I have spent some years both directly and indirectly in public health; my profession is essentially that of Dr. Salk, and I have been more or less familiar with federal government ways of standardization for 30 years.

The abysmally sad manner of handling the vaccine can be blamed on any one of six groups with complete success, in the same way that each of six cogs in a machine is the most important, since without any one the machine falls down. The seventh group, medicine, composed of men who, although not perfect judges, spend their lives in the specific and exact study of how to preserve health and diagnose and cure disease, might even so have been the deciding factor; but it was not even asked about the vaccine or any phase of the project.

These are the six troublesome cogs. Mr. O'Connor and his Infantile Foundation unquestionably stepped out of line in usurping unwarranted authority in deciding how to spend the money of other people, in choosing among various workers on vaccines which ones should be favored with hundreds of thousands of dollars and unlimited support, and in devising a circus out of what needed to be an evolutionary procedure of the greatest caution. He had stepped out of line before and it was someone's fault for not halting him.

Next, Dr. Salk, whose name is usually by-passed because, in common with many, his motives are not impugned, let himself become a pawn of Mr. O'Connor, relative to acceptance of funds, publicity and plan of operation. This act was only one of hundreds in modern science, but as yet no one has seen fit or had the courage to call for refusals of cash and glory in order to uphold some of the higher potentialities of man.

Third, public health crews far and wide—although by no means all of them—hopped on the bandwagon as soon as they saw it. They began propaganda long before even any figures were available, and helped in scores of ways to build themselves and the machine without the slightest knowledge of what it was all about. This propaganda and blatant optimism was and is wholly inexcusable.

Fourth, the commercial interests, six large companies, started in on a long gamble. It may perhaps be left to businessmen to decide how justifiable this gamble was in a business sense, but the facts indicate a combination of three poor decisions, succumbing to greed (which is legitimate business in its way), perhaps to the offers of Mr. O'Connor, whose temptations in terms of cash were terrific and whose prospects as a press agent were obvious, and to the shortsightedness of Dr. Salk and the federal government laboratories, neither of them as experienced or as expert as the commercial laboratories themselves.

Fifth, we have the fourth estate, the press, which failed to uphold its traditional honor of cold, tough skepticism. Faced with Mr. O'Connor, Dr. Salk, garbled figures, public health press agentry and the heavy commercial gamble of millions, the newspapers gave in and joined the fun. There was hardly a skeptical word, in the run-of-the-mill press, at least, although it is now evident that occasional individuals issued guarded statements to indicate that they were not entirely fooled. It is the press, with radio and TV, of course, which carries so many innocent bystanders along with it.

Sixth and finally, there is the federal laboratory group, among whom the technical men were hardly mentioned, and the higher-ups, legitimately unable to tell a vaccine from a serum, were made the goats, as usual. The fact is that men of these laboratories, including the wise men along with politicians and expedient artists, were also pulled into a part in the grandstand play instead of standing on their own feet. They lacked the independence to take a strong stand against it. The world of Salk, public health and grants was common experience to them, whereas the world of medicine is all too rarely heard in the realm of vaccines.

Because each of these has leaned heavily on the others, it is improper to lay the blame on any one, possibly excepting the instigator, but when all are included censure can be rather heavy.

Turning now to the problem itself, the usual terms are inadequate. The usual terms pretend to

Submitted August 23, 1955.

weigh the vaccine itself and the results secured with it. The vaccine is in no way original and is not really logically devised. It depended heavily on antibodies, although these have been produced by every vaccine ever invented, of which well over 90 per cent have failed. Furthermore, the use of formalin, known to be only a slowly lethal agent for microorganisms, especially the notably tough viruses, was bound to leave residual active virus. When the remaining virus became notably low, the fact that samples tested showed no virus failed to prove that vials not tested did not contain virus.

As for results, to many the "test of the pudding is in the eating." At least, so it seemed to many of the six groups and the innocent bystanders they were carrying with them. With this product, sensationally introduced, potentially dangerous and unproven, some of the points of truth were unquestionably seen by many medical men quite early. The opinions of these men were neither sought nor in many instances even allowed.

To get "results," seemingly so simple a matter, in 1954 the advance agents set up experiments with human subjects. The subjects were minors whose decisions were made for them by parents, a group of persons who might be expected to stand by their children but with whom fear played a tremendously influential role. The statisticians demanded "controls" and got them, although subsequent events proved, as was predictable, that controls were not only questionable experimentation but gave no aid in figures. The figures, whatever they were, were put in carefully palatable form by Dr. Thomas Francis, Jr., on April 12. How many have seen all the figures to weigh as they consider proper? The answer might satisfy some curiosity but would be of little consequence otherwise.

The answer to the layman, although not to the physician, seems absurdly simple. You simply vaccinate some children and turn them loose to see what happens. Then you add up the score to see whether or not the vaccinated children were protected. There is nothing to it. The child either gets poliomyelitis or he does not. Even a missed diagnosis would not matter much.

Simple as this looks, it requires a collection of observations because poliomyelitis is not common. The problem is not one of measuring benefits so much as one of measuring risks. Except for the circumstance of what was considered to be induced poliomyelitis in the spring of 1955, the risks would hardly have been mentioned. The bosses, who had the only files, admitted that there were in the 1954 experiments some 16 serious reactions and over 2500 minor reactions. Nothing much was said about the criteria of these reactions. Obviously it would be undiplomatic to make much point of any cases which occurred within a month after any of the injections, thus potentially induced, because this would reflect in retrospect on the whole chain, the bosses as well as the producers. In other words, in the experiment, we know little or nothing about the true total risks from the vaccine. Platitudes were murmured about some risks with all vaccines, and figures were produced such as that this year only 114 cases occurred in a short time after some 5,500,000 injections.

Examine the manner of that expression, designed to minimize the risk just as every figure favorable to the vaccine is presented to provide the greatest apparent benefit. These injections were given roughly during May, a low period for the disease. Newsmen are given figures saying that several hundreds of cases had occurred during May, not based on a comparable figure, 5,500,000. We are concerned in this country principally with about 32 per cent of some 160,000,000 persons, since for the most part the disease occurs among those under the age of 20. This is 51,200,000. Thus in round figures the number of cases among over 50,000,000 is set against the number of cases in more than 5,000,000. If the rate of poliomyelitis is about 10 per 100,000 in the population, over the normal year, it is around 30 per 100,000 for the 0-20 age group. This, however, is for the *year*. For a low month, May, the figure would be less than one-twelfth of 30, since August and September peaks may be 10 times higher than figures in other months. Suppose that there is usually one case per month per 100,000 persons under age 20, with ten cases per month in the two high months, for a first approximation. In short, some 55 cases might occur in May in 5,500,000 youths and young adults normally, but 114 are acknowledged. This hardly seems to reflect a minor accident, in the spirit of "a mere 114 in 5,500,000 and there are always some risks in vaccines anyway." This is the common abuse of apparent but not truly objective fact.

Consider another side of the risk, one which is fairly often discussed among medical men, although not often enough, and is otherwise kept almost entirely hidden. Are we so socialized that we must argue that saving five children at the expense of two who are killed who otherwise would have lived is a legitimate move? There are persons who so argue and who are even surprised that there is disagreement. All proper medical men and a large number of others will realize immediately that this outlook, whatever its social propriety, impropriety, or inhumanity, sets up a dictator, a man whose decisions are sacrosanct, the man who decides to kill some to save others.

Put it another way. Let us say that we can by vaccination kill two but save four. In other words,

the net gain is two; but two would be all that would have died anyway. Certainly it is not even arithmetically ethical under the circumstances to kill two and save four. And *who* will be allowed to make such decisions? Ethics of course demands that we not introduce risks, if there is any alternative, and certainly in the present problem there were plenty of alternatives.

Practitioners sometimes confuse this problem with those of their practices. Serious surgical operation or cortisone therapy, for instance, introduces pronounced risks which must be set against the chances of success. Medicine in this sense is a daily round of the calculated risk. These, however, are *patients*. These are persons for whom the decision is as to what shall be done in a case of trouble—vastly different from calling Johnny in from his baseball game to inject a product which may reduce as well as raise his batting average in future games with fate.

The figures which are tossed loosely about, including those so sensationally presented on April 12, vary from accidental to deliberate misleading data. The problem is one of relative risks rates, applied to a segment of one-third of the population. Humane considerations demand that natural risks must be accepted when induced risks are significant. Three groups, not two, must be weighed: Risks among those not vaccinated, risks among those vaccinated which have been added by the inoculation, and risks of the disease among those vaccinated. Since the last two are in opposition, it is easy to see why many unused to this subject accept a favorable balance without demand that the two risks be differentiated.

These groups fit about 32 per cent of the population. Furthermore, an important point, our concern is with *rates*. A rate is a unit of illness per unit of *time*, and nearly all the figures bandy time around as though it did not exist. In San Francisco 32 per cent of 775,000 is 248,000. From April 1 to August 19 there had been reported eight cases, or 1:31,000 under the age of 20. This is four and a half months, however, which puts a different aspect on it; and it makes a difference which four and a half months, or any period, is used.

Furthermore, no criteria of disease are acceptable in this deliberately induced scramble. What do we do about criteria with a disease which is regarded as beneficial if it occurs too lightly to be diagnosed, as of little consequence if it is recognized but does not paralyze, as dramatically serious if it paralyzes but with a fair percentage of fair recoveries, and as fatal in all too many cases but so irregularly that death, the sharp criterion, is not a measure of results. There has been a tendency to settle for paralysis as the criterion, but it is neither widely accepted nor does it lend itself to good figuring. Add to this difficulty the aforementioned differences in intervals of time, which are so much a part of any figure on rates, plus the fact that there are now children who were given the vaccine last year, those who had one shot this year, and those who have had more than one shot, plus the fact that one city considers one age group, another another, and most of them measure public health or school groups without regard to private inoculations—add these factors and you have some of the bases for the scramble, put on purely objective grounds.

On top of this, all the evidence has for years indicated that the great factor in immunity to this disease is in unrecognized infections. These are presumed to be some 100-fold the number of recognized infections. This, compared to any figures presented in any light, to date, makes our press agents' dreams a bit rosy, to put it mildly. Natural method is uncharted but it is large and effective, with man buzzing around its edges.

Can the score be added, even tentatively, at this stage? Possibly an idea can be gained. The April figures, carefully presented in terms of startling percentages, instead of in terms of calculated risks (for the most part the average of 60+ per cent, 70+ per cent, and 80+ per cent, was taken as 90 per cent!) were sadly misleading but slightly favorable in their way. That is, the chances of getting the disease in the age groups considered were around 1:2100 during the "season," whatever that was, and this was potentially reduced by the intensive series of inoculations to 1:3700. This is based on this age group, one season, intensive shooting, a single vaccine, and recognized cases. How to get even 60 per cent out of this is puzzling, but it indicates two things clearly, that at public health prices (with overhead discarded) an investment of $10,000 and up —probably well up—might save a case of poliomyelitis. It overlooks the fact that figures of this magnitude might do quite a bit in a number of directions, and it also fails to recognize that, when thousands must be vaccinated to protect one, such seemingly minor indirect risks as broken needles or various accidents, let alone possible induced infection or another disease superimposed on a reaction, become significant. To choose maximum benefits and ignore the balance, and to choose only direct damage and not total damage, is stacking the cards to the limit.

On an intuitive basis, the game does not seem to be worth the candle. The evidence does suggest some transitory immunity. Presumably many (certainly some) physicians have not used it on their own families, simply because the risk element and benefit values are both present and are both low. Physicians for themselves know the answer to that portentous question faced now by many parents: Is my con-

science eased more by having "done everything I could," even though I run into a trouble I might have avoided, or is it eased more by letting well enough alone and gambling on a kind fate which, if unkind, will leave me feeling that "conceivably I might have done so-and-so?" Most physicians, I surmise, know that fate is capricious. I surmise that they will and do take action against it hourly, but that they prefer a capricious fate to meddling when the gambling is really tough. And, incidentally, how would the minor himself vote? It is his life, not his parents' or physician's.

At the end of the season, plus a few months, we shall face a series of Aprils again. There will be tables, figures, and arguments. A majority will be for optimism, from mild to blatant. Great hopes will be expressed. Bromidic phrases about conquests and the greatness of science and billions for research will all be put forth. Few will mention that a large majority of vaccines in history for good and sufficient reasons have failed, but there will be plenty of platitudes about new, bigger, and better vaccines. Few of the figures will be meaningful, many will be unintelligible, and hardly any will consider true risk rates: Total damage and total benefit, not the difference between the two. The tremendous numbers of persons who must be inoculated to produce any good, even hypothetical, will be carefully hidden. Another season and presumably the dust will have settled a bit and perhaps evolution can take over what revolution messed up so beautifully. My own expectation is that another five years will see the vaccine gone, but this could be wrong, because, among other reasons, not every failure among vaccines has lost its supporters.

It might be pertinent, in closing, to ask whether anyone has learned anything. Considering the manner of propaganda, how much the public has learned is decidedly dubious. It might even be more gullible another time. In many ways Mr. O'Connor has shown himself to be irked—hardly a penitent attitude. Dr. Salk may not get far in terms of vaccine, but his fellow scientists go ahead to prove a multiplicity of Coxsackie viruses which resemble the poliomyelitis virus in numerous ways, and to demonstrate that there is apparently no such thing as adequately pooling even poliomyelitis viruses for either dead or live vaccine, thus getting a little closer gradually to the essential facts, out of which might eventually come something truly safe and mildly useful, if carefully introduced, slowly, and through the proper channels. There is no such assurance, however. Commercial interests are much chastened, but public health shows itself perhaps as ready as ever to catch on the passing calliope. A good many men in the news business have certainly learned a bit, but how widely effective this learning will become seems doubtful. Uncle Sam tangled up his politicians, who are only samples of the public in this technical matter, in the general confusion with the usual political repercussions; but possibly behind the scenes some of the men in the game have learned that they can afford to be a little independent, if honest, and do not have to follow the expedient path every time. This is not assured, either. By and large, the score perhaps is not without profit—a small one.

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California MEDICAL ASSOCIATION

NOTICES & REPORTS

Executive Committee Minutes

Tentative Draft: Minutes of the 252nd Meeting of the Executive Committee, San Francisco, Sir Francis Drake Hotel, September 14, 1955.

The meeting was called to order by Chairman Heron in the Cypress Room of the Sir Francis Drake Hotel, San Francisco, on Wednesday, September 14, 1955, at 6:30 p.m.

Present were President Shipman, Speaker Doyle, Council Chairman Lum, Auditing Committee Chairman Heron and Editor Wilbur. Absent for cause, President-Elect Charnock and Secretary Daniels.

A quorum present and acting.

Present by invitation were Messrs. Hunton, Clancy and Gillette of C.M.A. staff; legal counsel Hassard; health insurance consultant Waterson; Drs. Fred O. Cooley and Charles S. Mitchell of Fresno; Dr. John R. Upton, chairman of the Blood Bank Committee.

1. *Central California Blood Bank:*

Drs. Upton, Cooley and Mitchell reviewed the history of the Central California Blood Bank and discussed the operating problems encountered since opening of the institution on June 8, 1955. To date, they reported, the bank has not had sufficient support from various hospitals in Fresno and surrounding counties to permit an efficient operation.

On motion duly made and seconded, it was voted to extend further credit up to $20,000, in the form of a drawing account and with the understanding that the cooperation of at least one Fresno hospital is secured.

On motion duly made and seconded, it was voted to communicate with all Association members in the five-county area, urging their support of Central California Blood Bank.

On motion duly made and seconded, it was voted that the committee go on record as supporting a system of exclusive reciprocity for the Central California Blood Bank in its area; further, that the Blood Bank System, its respective member blood banks and their respective county medical societies support such exclusive reciprocity with Central California Blood Bank.

2. *Rollen Waterson Associates:*

Mr. Waterson discussed the study which he had proposed at an earlier meeting for a review of physician-patient relationships. The study would be designed to establish those areas where improvements might be accomplished through follow-up public relations activities. The study would be made by Stanford Research Associates of Palo Alto.

On motion duly made and seconded, it was voted to approve the making of this study at a cost not to exceed $5,000.

3. *Association of District Hospital Directors:*

Dr. Shipman discussed an invitation he had received, asking the Association to name a committee to meet with representatives of the Association of District Hospital Directors. It was agreed that he accept this invitation, provided the California Hospital Association were also represented.

4. *State Bureau of Vocational Rehabilitation:*

Dr. Shipman reported that some of the voluntary health agencies were taking an interest in the growing program of the State Bureau of Voca-

tional Rehabilitation, especially in view of some of the problems raised by the medical program of the bureau. On motion duly made and seconded, it was voted to authorize the President and the Council Chairman to appoint a committee to review the medical setup of this agency and report back.

Adjournment:

There being no further business to come before it, the meeting adjourned at 10:45.

IVAN C. HERON, M.D., *Chairman*
JOHN HUNTON, *Acting Secretary*

In Memoriam

BOYERS, LUTHER M. Died in San Francisco, September 19, 1955, aged 67, of myocardial infarction. Graduate of the Stanford University School of Medicine, Stanford University-San Francisco, 1916. Licensed in California in 1916. Doctor Boyers was a member of the Alameda-Contra Costa Medical Association.

✤

CRABTREE, PETER. Died in San Diego, September 8, 1955, aged 42, of carcinoma of the kidney. Graduate of the University of Michigan Medical School, Ann Arbor, 1937. Licensed in California in 1941. Doctor Crabtree was a member of the San Diego County Medical Society.

✤

HEANEY, N. SPROAT. Died in Los Angeles, September 25, 1955, aged 75. Graduate of Rush Medical College, Chicago, Illinois, 1904. Licensed in California in 1944. Doctor Heaney was a member of the Los Angeles County Medical Association.

✤

LOW-BEER, BERTRAM V. A. Died in Larkspur, September 25, 1955, aged 55. Graduate of Deutsche Universitat Medizinische Fakultat, Prague, Czechoslovakia, 1924. Licensed in California in 1943. Doctor Low-Beer was a member of the San Francisco Medical Society.

ROBERTS, WILLIAM H. Died in La Jolla, September 19, 1955, aged 83. Graduate of the Cooper Medical College, San Francisco, 1894. Licensed in California in 1894. Doctor Roberts was a retired member of the Los Angeles County Medical Association, the California Medical Association, and an associate member of the American Medical Association.

✤

SHRECK, JOHN A. Died in Redlands, September 3, 1955, aged 91. Graduate of Rush Medical College, Chicago, Illinois, 1891. Licensed in California in 1902. Doctor Shreck was a retired member of the San Bernardino County Medical Society, the California Medical Association, and an associate member of the American Medical Association.

✤

TORELL, GERHARD J. Died in Los Angeles, September 2, 1955, aged 62, of uremia. Graduate of Northwestern University Medical School, Chicago, Illinois, 1916. Licensed in California in 1924. Doctor Torell was a retired member of the Los Angeles County Medical Association, the California Medical Association, and an associate member of the American Medical Association.

✤

WAHRHAFTIG, MYER J. Died in Oakland, September 15, 1955, aged 67, of subarachnoid hemorrhage. Graduate of the Oakland College of Medicine and Surgeons, 1917. Licensed in California in 1917. Doctor Wahrhaftig was a member of the Alameda-Contra Costa Medical Association.

memo from: medical review and advisory board OF THE CALIFORNIA MEDICAL ASSOCIATION

What Price Medical Malpractice Insurance?

JOSEPH F. SADUSK, JR., M.D., Oakland

THE PROBLEMS associated with medical malpractice insurance are obvious to any physician, what with the rapid and progressive increase in premiums for such insurance during the past ten years. Articles on the subject have appeared in medical journals and other types of publications; some of these articles are sound, others reveal that the individual writing the article has little or no comprehension of the basic factors involved in this field.

The purpose of the present article is to discuss in general fashion the various factors playing a role in insurance coverage of this type. This article is written from physician to physician; therefore the details will be presented along the lines of interest to physicians rather than from the viewpoint of attorneys or insurance experts.

It is, first, important to realize that this problem is not confined to California. Indeed, the problem is nationwide, with particular emphasis recently in the states of New York, Illinois, Florida, Connecticut, Maryland and the District of Columbia. In some of these states the insurance carrier covering a group program has cancelled its group coverage because of increasing hazard in which the ratio of losses to premiums (loss ratio) continued to increase despite the rapid increase in premiums.

As an example of the increasing hazard in California, statistics show that for a group Northern California malpractice program during the period 1946 through 1951, one of every twelve physicians had a malpractice claim of some type levied against him each year. Breaking this figure down further, it is noted that one of every fifty-two physicians in the program had an actual malpractice suit filed against him each year, while one of every fourteen physicians had a serious allegation presented by a patient that required investigation by the insurance carrier. As a result, premiums in this group program have risen almost 200 per cent during the eight-year period from 1946 to 1954.

The growing dissatisfaction of insurance carriers with the field of medical malpractice insurance is disturbing. More and more carriers are dropping out of this type of insurance. Some carriers, realizing that this has been a losing field, are only offering the insurance to a physician if he permits the carrier to sell him other insurance such as automobile insurance, home insurance, personal liability insurance and so forth. This is commonly known as the "package deal."

DISSATISFACTION OF INSURANCE CARRIERS

Factors that make the insurance carrier dissatisfied may generally be classified as follows: (1) The problem of latent liability; (2) The progressive in-

• **The Medical Review and Advisory Board has been established as a committee of the Commission on Professional Welfare of the California Medical Association to make studies and recommendations toward solution of the growing problems of professional liability insurance and malpractice actions in California. The members of the Board are: Joseph F. Sadusk, Jr., Oakland, Chairman; Wilbur Bailey, M.D., Los Angeles, vice-chairman; Howard W. Bosworth, M.D., Los Angeles; H. I. Burtness, M.D., Santa Barbara; Paul W. Frame, Jr., M.D., Sacramento; Verne G. Ghormley, M.D., Fresno; Carl M. Hadley, M.D., San Bernardino; Joseph J. O'Hara, M.D., San Diego; William F. Quinn, M.D., Los Angeles; Rees B. Rees, M.D., San Francisco; and Bernard Silber, M.D., Redwood City; Mr. Rollen Waterson, 564 Market Street, San Francisco 4, is executive secretary, and Mr. Howard Hassard is legal counsel.**

crease in losses in such insurance due to inflation and the increasingly critical attitude of the public, the juries and the courts; (3) The small volume of sales of this type of insurance; and (4) The growing dissatisfaction of physicians with the progressive increase of premiums by means of which the insurance carrier hopes to balance the program.

The latent liability factor is entailed in the long lapse of time between the alleged act of malpractice and to filing of a claim or suit. With automobile liability insurance, the carrier knows at the end of the given policy year or shortly thereafter the entire extent of its liability. With medical malpractice, however, claims for alleged malpractice may come up many years after the incident that is cited as a basis for claim. This is due to the very unfavorable statute of limitations in California which basically provides that a patient may file a suit or claim against a physician one year after the patient has acquired knowledge of an act of malpractice. Basically, this means that the patient has practically his entire lifetime or the physician's lifetime in which to file suit. For instance, if a surgeon inadvertently leaves a clamp in an abdomen during an operation and the patient is told forty years later when a gastrointestinal series is made that such a clamp is present, the patient has one year thereafter to file a suit. Consequently, it is extraordinarily difficult for insurance carriers to predict losses with any accuracy for a given policy year, since the policy for the year in which the incident occurs is the policy which covers the physician for the rest of his life, regardless of the year in which the suit is filed.

That both the incidence of malpractice claims and size of judgments and settlement costs are increasing is clear not only in California but in other states. For instance, actuarial data in one state reveal that the incidence of malpractice suits per unit number of physicians has increased 100 per cent during the past ten years. Inflation has likewise produced an increase in judgments and an increase in the cost of defense during the same period.

In contrast with other types of insurance, sales in malpractice insurance are relatively small and consequently an insurance carrier would make relatively little profit, even if this type of insurance were profitable. As a result, the average insurance carrier looks upon the selling of malpractice insurance as a "courtesy" or "accommodation" line, rather than as a profitable enterprise.

Another facet of the problem is the growing dissatisfaction of physicians with increasing premium rates. This dissatisfaction is due to the failure of the physician not only to realize the problems involved, but also the fact that such insurance has recently been a losing proposition to the carrier. The Medical Review and Advisory Board has had the opportunity of reviewing financial data for a number of insurance carriers selling malpractice insurance in California. In no instance was the board able to find evidence of even a reasonable profit; indeed, the carriers making information available to the board presented statistics which showed that the insurance coverage was carried at a financial loss during the period studied.

CALCULATION OF PREMIUMS

Medical malpractice insurance premiums, like premiums for other types of insurance, are calculated on the basis of expected losses plus expected expenses. In the best of circumstances where accurate data are available on the history of losses, the insurance carrier takes this financial data into account and adds to it the cost of administering the policy (sales, federal and state taxes, home and district office expenses, and employees' salaries), costs of special investigation, court costs, attorneys' fees, and agents or brokers' commissions (if the company is a stock company) to arrive at a given annual premium. In the case of a stock company, a fair dividend return to the stockholders is included in the expenses. In the case of a mutual company, any profit resulting from a given year's sale of the insurance returns to the buyer either in the form of a dividend or as reduced premiums in future years. In the case of medical malpractice insurance, the calculation of premiums is extraordinarily difficult due to the factors of latent liability, inflation, an increasingly critical attitude of the public, and the generosity of juries in awarding higher and higher judgments. Therefore, medical malpractice premiums are set by an educated guess at the very best in this day of rapidly increasing problems.

"RESERVES"

Many physicians have asked about the question of "reserves." There are generally two types of reserves in any insurance company. First come the general reserves of the company which are generally set aside for catastrophic events and generally depend upon the size of the insurance carrier. Second, are the reserves that are set aside in safe-keeping when a case with possible loss is reported, so that there is a guarantee to the holder of the insurance policy that there will be sufficient money to pay the claim, or judgment in a suit, if and when such payment becomes due. This type of reserve is set up by the company and represents, in the best judgment of the insurance carrier, the amount that the claim or suit will cost, taking into consideration the award to the plaintiff, court costs, attorneys' costs in defending, and costs of special investigation.

Such reserves may vary in amount from less than $100 to even the entire limit of coverage of the

physician, depending upon the seriousness and liability of the case. If there is no liability, the reserves will be very minimal, reflecting only the costs of special investigation.

In general, insurance carriers are very skillful in setting up such reserves and in most instances successful claims over a period of one or several years when averaged out for many cases come to within 10 or 15 per cent of the amount originally set aside. When money is set aside in such specific reserves, the money is not necessarily lost to the program. In other words, if the case is successfully consummated in so far as the defendant physician is concerned, the money is taken out of the reserve and is put back into the program, thereby eventually lowering premiums or becoming payable in the form of dividends.

MUTUAL vs. STOCK COMPANIES

There has been much discussion concerning pros and cons of the so-called mutual company versus the stock company. In general, a stock insurance carrier is owned by shareholders, such as any ordinary corporation. These shareholders receive a dividend return on their shares of stock, ranging from two to five per cent, generally. With a mutual company, there are no stockholders; indeed, the policyholders themselves own the company by virtue of their policies. There are no stockholders to pay, and any profits resulting at the end of the year are added to general surplus or returned to the policy holder by the payment of dividends or reduction of premiums during the next year. Also, in general, policies for stock companies are sold through agents or brokers, while policies for certain mutual companies are transacted by salaried company employees.

It is profitless to discuss the pros and cons of the stock versus mutual company, since the matter is one of individual preference. Further, the efficiency, skill, and service of companies is a variable factor. The important point is that the policyholder have his insurance in a reputable, long-established company which will give every assurance of being in the business throughout the lifetime of the physician and that has assets in the United States available for defense and paying claims. Whether the company be stock or mutual is of little concern, and premiums are of secondary consideration.

The important criteria for a physician to consider in selecting a carrier are:

1. Past and future stability of carrier.

2. Adequate reserves of the carrier on deposit in the United States.

3. Group program sponsored and monitored by a county or state medical society.

4. Offering by the carrier of sufficiently high coverage to adequately meet a high judgment.

5. Limitation of "cancellation" clause.

6. Limitation of "exclusion" clauses.

7. Contingent liability coverage (for the acts of a physician's partner).

8. Permanence for yearly renewal of the policy.

9. Absence of hidden additional charges.

10. Sufficient volume of business in the area in which the physician practices to have experienced malpractice claims adjustors and defense counsel.

GROUP PROGRAM vs. INDIVIDUAL POLICIES

There is little or no doubt that a group malpractice program has many advantages over the individual malpractice policy. Here the physician has the basic advantage of group protection and group negotiation with the carrier along the following lines:

1. Prevention of discrimination against physicians whom the carrier considers hazardous risks because of their field of work. (This danger already exists. For instance, there is one insurance carrier which rejects orthopedists, plastic surgeons, and radiologists from coverage.) Who is to be excluded from medical malpractice coverage should be a determination of physicians rather than insurance people.

2. Prevention of the possible control by the insurance company over what procedures the doctor may perform. Such control, preventing the performance of hazardous procedures, may easily be accomplished by the carrier through exclusions in the individual policy or by the device of surcharges so high the physician cannot afford to pay the premium.

3. The medical profession itself may have considerable influence over which claims are to be settled and which are to be defended in a group program. Thus the decision is influenced by analysis of the merit of the claim rather than by expediency.

4. The training of competent claims managers and the team approach to claim handling and defense is accomplished in a group program. Such effective programs are found in groups rather than in individually handled policies.

5. An effective prevention—or safety—program may be set up with a long-term view of reducing the incidence of claims or lessening liability.

6. The collection of loss data by physicians in a group program may be accomplished with the objective of testing the reasonableness of premiums and, most important, of establishing the underlying or real causes of claims.

7. The long-range interests of the profession are served in a group program rather than the immediate

interests of the insurance carrier by physicians influencing and guiding the course of malpractice insurance. The insuror can desert the field of malpractice, but the profession has to live with it. The problem is basically medical in nature rather than insurance.

LOCAL GROUP PROGRAMS vs. NATIONAL GROUP PROGRAMS

What has been said above on the subject of group programs in malpractice insurance does not necessarily apply to national specialty group programs. Several such national programs have been started in the recent past, and one such specialty group already has been suddenly dropped by the insurance carrier involved.

While a group national specialty program may have a cost advantage over individual insurance, this has not yet been demonstrated. On the other hand, national programs are not as advantageous to the physician as a local group program. The principal disadvantage of a national program is that it may lack concentration in an area, and therefore have so few claims that it does not have claims adjustors and attorneys sufficiently well qualified in the field. The handling of medical malpractice problems by the underwriters of national programs may be a very minor part of their total general work.

Another difficulty with the national program is the remoteness of the central claims office from the field of action. Consequently, there may be a tendency on the part of the carrier to settle on the basis of expediency, medical-professional appraisal of the case with coordination of the claims adjustor may be poor, and there may be little or no contact of the physician with the carrier.

Still another problem with national groups is the difficulty in setting up prevention or safety programs because of local state differences, court procedures and legal codes. Malpractice is not within the province of federal courts, but rather is governed by local and state courts—with differences not only among states but also among counties.

AMOUNT OF COVERAGE

With respect to the amount of insurance coverage for medical malpractice, several articles have lately appeared in journals circulated to physicians, recommending low coverage. The argument is that low coverage will discourage claims and will tend to lower the amount of plea for damages or settlement. The author has never known or heard of a single malpractice case in which the plea for damages or settlement was influenced by the amount of insurance coverage. On the contrary, physicians leave themselves open to financial ruin with low coverage.

The problems connected with inadequate coverage have been increasingly great during the past several years. Recently in California two judgments in excess of $200,000 each were rendered. The author has been closely associated with the field of malpractice during the past five years and has seen the near tragedies which result with low coverage. The physician who carries coverage of $5,000/15,000 or $10,000/30,000 is indeed an unhappy person when he is faced with a suit in which the plea may be up towards $200,000, and if damages are awarded, the judgment may well run in the neighborhood of $50,000 to $75,000. The author has seen physicians become almost psychotic persons with the worry and fear of the approaching trial in cases of that kind.

What is considered adequate coverage? The answer is difficult, but of course basically it depends upon the individual's assets, both present and in prospect, to be protected, and also upon his type of practice. In general, a coverage of less than $50,000/100,000 should be considered inadequate in these days, and generally it is well to have coverage of $100,000/300,000. For physicians engaged in particularly hazardous work, such as anesthesiologists, vascular surgeons and neurosurgeons who perform work of a type that, regardless of skill and care, may lead to paraplegia as a complication, coverages up to $300,000/900,000 may be considered desirable. This is based specifically upon the fact that in California within the past few months, verdicts of $225,000 and $250,000 respectively have been awarded by juries for paraplegia following spinal anesthesia in one case and aortogram in another. The day of the $5,000/15,000 and $10,000/30,000 coverage has passed.

THE FUTURE

The future for the physician in medical malpractice insurance is dismal indeed. Each year brings forth new medical discoveries of importance with benefit to the patient; but as medicine progresses so likewise difficulties increase in practice with the use of complicated surgical procedures and the administration of toxic drugs. These lead necessarily to an irreducible number of complications for which the physician may be held responsible, and likewise lead to a greater burden placed upon the physician by the courts and by an increasingly critical public. The great advances in medicine and surgery, as presented to the layman in magazines, have led the public to be supercritical in appraising results. Our grandfathers didn't expect the horse-and-buggy doctor to be perfect, but our contemporaries expect perfection of today's physicians.

To seek aid from the public, the attorney, the legislature, and the courts is not necessarily the

answer at this time. The physician must begin the battle himself. As Ford[1] pointed out five years ago, in probably the shortest paper ever published on the subject but nevertheless most revealing, the control of medical malpractice hazards depends upon:

Good faith
Good records
Common sense

Good faith implies that the physician treat his patient with tact and kindness, that he conceal no known difficulty in diagnosis or treatment, and that he advise consultation freely.

Good records means that the physician adequately document his medical records of a patient, carefully record untoward happenings, and make a matter of record the treatment given and advice offered.

Common sense implies that the physician know the vindictiveness of some patients, recognize the hazard connected with the collection of reluctant fees, be aware of the failure of equipment which in turn can produce injury, and finally, use only well established medications and procedures.

REFERENCE

1. Ford, R.: Medical malpractice, New Eng. J. Med., 243:408, Sept. 14, 1950.

CANCER DIAGNOSTIC TESTS: *PENN REACTION*

A Statement by the Cancer Commission of the California Medical Association (May 1954; reaffirmed, October 1955.)

FROM TIME TO TIME, announcements appear in the press concerning new alleged cancer tests. Up to the time of preparation of this statement, not one of the numerous "blood tests" for cancer has withstood scientific investigation. Many have given rise to false positive results with distressing consequences to patients and their families.

During the last few months, there has been considerable publicity concerning a so-called seroflocculation reaction for cancer, otherwise known as a Penn or Penn-Dowdy blood test. As far as the Cancer Commission of the California Medical Association can ascertain, the following is the present status of this procedure.

1. The Penn seroflocculation reaction is not a cancer test. It is positive in a majority of patients with cancer *and* in patients who:

(a) recently have had injury or operation;
(b) have active rheumatoid arthritis;
(c) have cirrhosis of the liver;
(d) have fever over 100 degrees;
(e) have active tuberculosis;
(f) are pregnant;
(g) are taking medication such as desiccated thyroid, estrogens, insulin, epinephrine and corticotropin (ACTH).

In other words, this experimental test is positive in many conditions besides cancer, and is therefore nonspecific.

2. In a certain number of patients who actually have cancer, the reaction is negative. The precise number of such false negative reactions and of the previously mentioned false positive reactions is under investigation at present. It will take many months, if not years, to complete this investigation. The minute that reliable information concerning the value of this reaction in independent hands is available, it will be made public.

3. Should this reaction prove to be of such value as to endorse its general use, it would constitute a supplementary item of evidence in the differential diagnosis of cancer. Its responsible proponents do not suggest, as yet, that it deserves any consideration in mass screening of asymptomatic individuals.

4. The National Research Council maintains a Committee on Cancer Diagnosis and Therapy. This committee has prepared criteria for the evaluation of diagnostic procedures. The Cancer Commission of the California Medical Association has recommended that investigators note these carefully prepared criteria and that due attention be given to them in making clinical tests on any type of proposed cancer diagnostic procedures.

5. Pending the discovery of a particular blood or chemical test, citizens are urged to utilize tried and tested methods of cancer detection. The most reliable method consists in physical examination by a qualified physician.

Annual Meeting

Ambassador Hotel

LOS ANGELES

April 29 - May 2, 1956

Papers for Presentation

If you have a paper that you would like to have considered for presentation, it should be submitted *to the appropriate section secretary* (see list on this page) not later than November 19, 1955.

Scientific Exhibits

Space is available for scientific exhibits. If you would like to present an exhibit, please write immediately to the office of the California Medical Association, 450 Sutter Street, San Francisco 8, for application forms. To be given consideration by the Committee on Scientific Work, the forms, completely filled out, must be in the office of the California Medical Association not later than December 1, 1955. (No exhibit shown in 1955, and no individual who had an exhibit at the 1955 session, will be eligible until 1957.)

SCIENTIFIC PAPERS

SCIENTIFIC EXHIBITS

PLANNING MAKES PERFECT

AN EARLY START HELPS

SECRETARIES OF SCIENTIFIC SECTIONS

ALLERGY . . . . . **William J. Kerr, Jr.**
711 D Street, San Rafael

ANESTHESIOLOGY . . . . . **Robert W. Churchill**
1180 Montgomery Drive, Santa Rosa

DERMATOLOGY AND SYPHILOLOGY . . **Anker K. Jensen**
1052 West Sixth Street, Los Angeles 17

EAR, NOSE AND THROAT . . . . **E. Gordon McCoy**
490 Post Street, San Francisco 2

EYE . . . . . **Channing W. Hale**
174 Nemaha Street, Pomona

GENERAL MEDICINE . . . . . **Harold C. Sox**
300 Homer Avenue, Palo Alto

GENERAL PRACTICE . . . . . **T. Jackson Laughlin**
10910 Riverside Drive, North Hollywood

GENERAL SURGERY . . . . . **Orville F. Grimes**
U. C. Medical Center, San Francisco 22

INDUSTRIAL MEDICINE AND SURGERY . . . . . **Homer S. Elmquist**
629 So. Westlake Avenue, Los Angeles 57

OBSTETRICS AND GYNECOLOGY . . . **Ralph C. Benson**
U. C. Medical Center, San Francisco 22

ORTHOPEDICS . . . . **A. B. Sirbu (Acting Secretary)**
450 Sutter Street, San Francisco 8

PATHOLOGY AND BACTERIOLOGY . . **Justin R. Dergeloh**
378 Thirtieth Street, Oakland 9

PEDIATRICS . . . . . **Moses Grossman**
U. C. Medical Center, San Francisco 22

PSYCHIATRY AND NEUROLOGY . **William F. Northrup, Jr.**
696 East Colorado Street, Pasadena 1

PUBLIC HEALTH . . . . . **Wilber J. Menke, Jr.**
City Hall, Pasadena 1

RADIOLOGY . . . . . **Austin R. Wilson**
540 North Central Avenue, Glendale 3

LEGISLATION . . . role of the physician's wife

Perhaps nowhere are the complexities and pressures of our present-day American civilization reflected more vividly than in the field of legislation. On local, state and national levels there are tens of thousands of bills introduced at each session of our various legislative bodies. These bills reflect the needs and "special interests" of countless groups. Many of these bills are excellent and necessary; many are potentially harmful. All of them are of interest and concern to all citizens. And some of them are of vital importance to the medical profession.

How can the busy physician possibly keep abreast of this current legislation? It is obviously beyond his scope to read and interpret the 11,914 bills, for instance, that were introduced in the first session of the 84th Congress—or the 6,227 measures introduced into the California State Legislature. It is even a colossal task to read through the 404 Congressional bills that were considered of special importance to the medical profession. Physicians have a legislative study group (the Public Health League) to sift out significant legislation and to keep them well informed and alert to the current legislative problems. This is of prime importance. Of equal importance is that the wives of physicians be well informed and alert.

Indeed, a physician's wife has a double responsibility to keep aware of the complex and changing legislative picture. Not only can she be of immeasurable help in keeping her husband up to date, she can also supply valuable information to her friends and neighbors. Far too many people are in the dark where medical legislation is concerned, and a well informed Auxiliary member is often the one who can present all sides of the picture most effectively.

In recognition of this, your Woman's Auxiliary incorporated a Legislative Committee into its original program. This committee works hand in hand with the legislative committees of the county societies, the C.M.A. and the A.M.A., thus receiving its working information on the broadest possible scale. It also keys its program with that of the legislative committee of the Woman's Auxiliary to the A.M.A. and works with and through the county auxiliaries in disseminating information and formulating local programs.

Keeping abreast of current legislation calls for eternal vigilance, and your Auxiliary has been active almost continually since its inception in 1929. Some years, however, have called forth more significant efforts than others. In 1930 and 1931, for instance, the Auxiliary made an appraisal (working with Mr. Ben Read) of California health laws to acquaint its membership with their workings. In 1936 and 1937, when a threat of socialized medicine appeared, your Auxiliary informed its members of the various aspects of socialized medicine so that they could convey effectively to the public accurate information concerning all that was involved. The following year, the Legislative Committee was active in helping to defeat the Humane Pound Act proposed by the anti-vivisectionists. In 1948, the Auxiliary again recorded its opposition to socialized medicine. By 1951, our legislative study groups were gathering momentum toward having all members well informed politically.

Our more recent activities have been numerous and complex. Aside from furnishing the pros and cons of current important bills to our members, we have attempted to impress on them the importance of knowing who their representatives are and what they stand for. To this end, we have encouraged our members to acquaint themselves personally with their local candidates, to attend their local legislative sessions and to express their opinions to their representatives in letters, phone calls or telegrams.

The Auxiliary itself keeps in close touch with state legislative bodies. Last year, the California State Legislature, well acquainted with your Auxiliary, commended the Auxiliary (under the able leadership of Mrs. Frederick Miller) as an organization "which has rendered many valuable services throughout the state."

Your Auxiliary works constantly with the C.M.A. whenever an emergency arises. Through the use of letters, phone calls and telegrams when necessary, it makes its voice heard. Key "contact women" in every community stand ready to alert our members when speedy action is needed.

While the Auxiliary's legislative program gives emphasis to bills with primary medical implication, our legislative activities are by no means limited. One of our main objectives is to support good general legislation. To this end, we cooperate with many other interested community groups, such as the Parent-Teachers Association, legislative councils, and the League of Women Voters, in our study and planning. One of our important local activities lies in helping to "get the vote out." Auxiliary members have worked most successfully with local registration bureaus in setting up and staffing registration booths in medical buildings and in hospitals.

The Auxiliary legislative objectives call for the "promotion of legislation that will advance the kind of medical care beneficial to the health of all the people" as well as for opposition to "legislation with detrimental medical implications." On our study agenda for 1956 are the four subjects following: (1) Social Security—disability coverage; (2) the Jenkins Keogh Bills; (3) Reinsurance; (4) the Bricker Amendment. In studying the issues involved, we will attempt to apprise ourselves of *all* phases of the problems. We believe that only by considering the many sides of a controversial picture can we form an intelligent, unbiased opinion.

NEWS & NOTES

NATIONAL • STATE • COUNTY

LOS ANGELES

Dr. James B. Johnson of Beverly Hills was elected vice-president of the American Society of Plastic and Reconstructive Surgery at a meeting of the organization held recently at Atlantic City, New Jersey.

* * *

In cooperation with the American Psychiatric Association, the Department of Psychiatry of the U.C.L.A. School of Medicine and the University of California Extension have announced plans for a two-day western regional research symposium on the **"Application of Basic Science Techniques to Psychiatric Research,"** to be held in Los Angeles, January 26 and 27. The course will be open to psychiatrists, psychologists, psychiatric social workers, psychiatric nurses and others with the consent of the course chairman, according to Thomas H. Sternberg, M.D., assistant dean for postgraduate medical education at U.C.L.A. Requests for information and registration blanks should be addressed to Dr. Sternberg at the University of California School of Medicine, Los Angeles 24, California.

* * *

Dr. Lawrence H. Herman of Monterey Park, an instructor in cardiology at the University of Southern California School of Medicine, has been appointed an associate clinical professor at the institution.

* * *

The University of Southern California was among the recipients of grants recently announced by the **National Foundation for Infantile Paralysis** to aid in research and in training for personnel for therapy. The institution was allotted $19,092 to schedule courses for graduate and undergraduate occupational and physical therapy students in treatment of poliomyelitic patients, under direction, respectively, of Angeline A. Howard, chairman, department of occupational therapy, and Charlotte W. Anderson, chairman, department of physical therapy.

SAN BERNARDINO

Dr. Frank C. Melone of Ontario took office as president of the San Bernardino County Medical Society at the annual meeting of the society, held in October. He succeeded Dr. H. R. Morris of Redlands. Dr. Ben D. Miano of San Bernardino was elected president-elect. The new vice-president is Dr. Phillip Savage of San Bernardino. Dr. Wendell L. Ogden, of Highland, was elected secretary-treasurer.

GENERAL

At the annual meeting of the **State Board of Medical Examiners** in Sacramento, held October 20, 1955, the following officers were elected for 1956: President, Justin J. Stein, M.D., Los Angeles; vice-president, Clayton Mote, M.D., San Francisco, and secretary-treasurer, Louis E. Jones, M.D., Sacramento.

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The Tenth Annual University of Florida Midwinter **Seminar in Ophthalmology** and Otolaryngology will be held at the Sans Souci Hotel in Miami Beach the week of January 16-21, 1956. The lectures on ophthalmology will be presented on January 16, 17 and 18, and those on otolaryngology on January 19, 20 and 21. A midweek feature will be the Midwinter Convention of the Florida Society of Ophthalmology and Otolaryngology on Wednesday afternoon, January 18, to which all registrants are invited.

* * *

The Tenth Annual **Symposium on Fundamental Cancer Research** will be held on March 29, 30 and 31 at The University of Texas M.D. Anderson Hospital and Tumor Institute in the Texas Medical Center, Houston.

* * *

The U. S. Public Health Service recently announced ten grants totaling $295,367 to start a special program of **research into air pollution problems.** Three grants for a total of $62,141 were awarded to investigators in California: $10,982 to Paul Kotin, University of Southern California for determination of acute and subacute biologic effects of air pollutants; $36,784 to Hurley L. Motley, University of Southern California, for investigation of the effect of breathing smog air on pulmonary function in man, and $14,375 to Bernard D. Tebbens, University of California, Berkeley, for particulation of air pollutants resulting from combustion.

POSTGRADUATE EDUCATION NOTICES

THIS BULLETIN of the dates of postgraduate education assemblies and the meetings of various medical organizations in California is supplied by the Committee on Postgraduate Activities of the California Medical Association. In order that they may be listed here, please send communications relating to your future medical or surgical programs to: Mrs. Margaret H. Griffith, Assistant Director, Postgraduate Activities, California Medical Association, 417 South Hill Street, Los Angeles 13.

UNIVERSITY OF CALIFORNIA AT LOS ANGELES

Dermatology in General Practice, November 16 to December 21. Twelve hours. Fee: $30.00.

Electrocardiography, Wednesdays and Fridays, November 30 to December 16. Twelve hours. Fee: $20.00.

In Santa Barbara: New Diagnostic Procedures and Therapy in Medicine, Wednesday, November 30 (afternoon and evening). Fee: $17.50.

Dermatology 1956, Friday and Saturday, June 22 and 23, 1956. Twelve hours.

Contact: Thomas H. Sternberg, M.D., Assistant Dean for Postgraduate Medical Education, U.C.L.A., Los Angeles 24.

UNIVERSITY OF CALIFORNIA, SAN FRANCISCO

In San Francisco:

Ophthalmological Conference—December 5 to December 7.

Conference on Dermatology for General Practitioners, January 13 and 14, 1956. Twelve hours. Fee: $40.00.

In Santa Rosa: Symposium on Heart Disease, Wednesday, November 16. Seven hours. Fee: $12.50. Sponsored by Sonoma County Heart Association and in cooperation with Stanford University School of Medicine.

Contact: Seymour M. Farber, M.D., Head, Postgraduate Instruction, Office of Medical Extension, University of California Medical Center, San Francisco 22.

UNIVERSITY OF SOUTHERN CALIFORNIA, LOS ANGELES

In Los Angeles:

No. 834: Electrocardiography, Friday, Saturday and Sunday, February 10, 11, 12. All day. Fee: $50.00.

Contact: Phil R. Manning M.D., Director of Medical Extension Education, University of Southern California School of Medicine, 2025 Zonal Avenue, Los Angeles 33.

COLLEGE OF MEDICAL EVANGELISTS

Anesthesiology. Daily, full-time, four months, beginning each four months. Fee: $300.

Medical Clinics, Tuesdays, January 3 to March 20, 1956. Eighteen hours. Fee: $50.00.

Differential Diagnosis of Internal Diseases, Tuesdays, January 3 to March 20, 1956. Eighteen hours. Fee: $50.00.

General Urology, Wednesdays, January 4 to February 29, 1956. Thirteen and one-half hours. Fee: $35.00.

Management of Infertility, Thursday, January 5 to February 23, 1956. Twelve hours. Fee: $30.00.

Dermatology, Tuesdays, January 10 to March 27, 1956. Eighteen hours. Fee: $40.00.

Varicose Veins and Surgical Diseases of the Peripheral Vascular System, Tuesdays, January 17 to February 28, 1956. Fourteen hours. Fee: $30.00.

Otolaryngology, Tuesdays, February 7 to March 27, 1956. Twelve hours. Fee: $30.00.

Gynecology, Wednesdays, March 21 to May 23, 1956. Ten hours. Fee: $30.00.

Operative Surgery, Wednesdays, March 21 to June 6, 1956. Thirty hours. Fee: $200.00.

Thoracic Surgery, Wednesdays, April 18 to May 9, 1956. Eight hours. Fee: $30.00.

Diseases and Injuries of Bones and Joints, Daily, July 2 to July 31, 1956. Full time. Fee: $100.00.

Contact: Chairman, Section on Graduate and Postgraduate Medicine, College of Medical Evangelists, 1720 Brooklyn Ave., Los Angeles 33.

CALIFORNIA MEDICAL ASSOCIATION POSTGRADUATE INSTITUTES

SOUTHERN COUNTIES in association with the University of Southern California School of Medicine, January 19-20, 1956, at Laguna Beach.

WEST COAST COUNTIES in association with College of Medical Evangelists, March 1-2, 1956, in Carmel.

NORTH COAST COUNTIES in association with University of California School of Medicine, San Francisco, April 5 and 6, 1956, in Santa Rosa.

SAN JOAQUIN VALLEY COUNTIES in association with the University of California School of Medicine, Los Angeles, May 10 and 11, 1956, in Fresno.

SACRAMENTO VALLEY COUNTIES in association with Stanford University School of Medicine, June 21, 22, 1956, at Lake Tahoe.

Contact: C. A. Broaddus, M.D., Director of Postgraduate Activities, P.O. Box A-1, Carmel, California, or Mrs. Margaret H. Griffith, Assistant Director, Postgraduate Activities, California Medical Association, 417 So. Hill St., Los Angeles 13.

Medical Dates Bulletin

NOVEMBER MEETINGS

STOCKTON POSTGRADUATE STUDY CLUB, State Hospital, Stockton, November 17.

Contact: L. Armanino, M.D., 2633 Pacific Avenue, Stockton.

AMERICAN MEDICAL ASSOCIATION Clinical Session, 1955, in Boston, November 29 to December 2, 1955.

CITY OF HOPE MEDICAL CENTER, Newer Developments in the Diagnosis and Treatment of Cancer, November 28, 29, 30. All physicians invited to attend.

Contact: Leo G. Rigler, M.D., City of Hope Medical Center, Duarte, Calif.

MONTEREY COUNTY HEART ASSOCIATION Annual Symposium on Heart Disease, House of Four Winds, Monterey. Fee $10.00 for 2 days, $7.00 for one day, $5.00 for one-half day, November 30 and December 1.

Contact: Edwin W. Tucker, M.D., 1073 Cass Street, Monterey.

DECEMBER MEETINGS

MEDICAL ALUMNI COMMITTEE OF CHILDREN'S HOSPITAL, San Francisco, December 2, 9:30 a.m.* Prenatal Problems, Departments of Obstetrics, Anesthesia, Pediatrics and Public Health, participating.

REGIONAL CONFERENCE ON PHYSICIANS AND SCHOOLS, San Jose, December 2 and 3.

Contact: Talcott Bates, M.D., 920 Cass Street, Monterey.

CALIFORNIA HEART ASSOCIATION semiannual meeting, Hotel Statler, Los Angeles, December 3.

Contact: Alan Croft Blanchard, Field Director, 1428 Bush Street, San Francisco 9.

AMERICAN COLLEGE OF CHEST PHYSICIANS, Ambassador Hotel, Los Angeles, December 5-10. Los Angeles Postgraduate Course on Diseases of the Chest.

Contact: Alfred Goldman, M.D., Chairman, 416 North Bedford Drive, Beverly Hills.

JANUARY MEETINGS

LOS ANGELES MIDWINTER MEDICAL CONVENTION, January 3, 4, 5, 1956, Biltmore Hotel, Los Angeles. An 85th anniversary, sponsored by Los Angeles County Medical Association.

Contact: Jerry L. Pettis, Public Relations Counsel, Los Angeles County Medical Association, 1925 Wilshire Blvd., Los Angeles 57. Telephone DUnkirk 5-1581.

LOS ANGELES COUNTY MEDICAL ASSOCIATION "Cavalcade of Health and Medical Progress," Shrine Auditorium, Los Angeles, January 6 to 15.

Contact: Bert Fitzgerald, business manager, Los Angeles County Medical Association, 1925 Wilshire Boulevard, Los Angeles 57.

MEDICAL ALUMNI COMMITTEE OF CHILDREN'S HOSPITAL, San Francisco, January 14, 1956.* Pediatric Surgery, with special emphasis on diagnosis, referral, preparation, pre- and postoperative care.

FEBRUARY MEETINGS

ALAMEDA-CONTRA COSTA MEDICAL ASSOCIATION Graduate Assembly. "The Dynamics of Endocrine Disease," Highland-Alameda County Hospital, February 10.

Contact: L. W. Kinsell, M.D., Instructor for Metabolic Research, Highland-Alameda County Hospital, Oakland.

AMERICAN BOARD OF SURGERY EXAMINATIONS, Part II, Los Angeles, February 13 and 14. Closing date is December 1.†

AMERICAN BOARD OF SURGERY EXAMINATIONS, Part II, San Francisco, February 16 and 17. Closing date is December 1.†

SPRING MEETINGS

MEDICAL ALUMNI COMMITTEE OF CHILDREN'S HOSPITAL, San Francisco, March 17, 1956.* Morning: Dermatology. Afternoon: Nutritional problems peculiar to modern pediatrics.

CALIFORNIA TUBERCULOSIS AND HEALTH ASSOCIATION, California Trudeau Society and California Sanatorium Association Annual Meeting, Sheraton-Palace Hotel, San Francisco, April 5, 6, 7.
Contact: E. L. Daggett, director, Public Relations, California Tuberculosis and Health Association, 130 Hayes Street, San Francisco 2.

MEDICAL ALUMNI COMMITTEE OF CHILDREN'S HOSPITAL, San Francisco, April 14, 1956.* Behavior Problems and Childhood Psychiatry.

AMERICAN COLLEGE OF PHYSICIANS 37TH ANNUAL SESSION, Los Angeles, April 16-20, 1956.
Contact: George C. Griffith, M.D., General Chairman, Box 25, 1200 N. State St., Los Angeles 33.

HAWAII MEDICAL ASSOCIATION Centennial Celebration. Scientific sessions, historical pageant of 100 years of medicine in Hawaii, social festivities, etc., Honolulu, April 22 to 29.
Contact: Hawaii Medical Association, 510 S. Beretania Street, Honolulu 13, Hawaii.

CALIFORNIA MEDICAL ASSOCIATION ANNUAL MEETING, Ambassador Hotel, Los Angeles, April 29 to May 2, 1956.
Contact: John Hunton, Executive Secretary, 450 Sutter St., San Francisco 8, or Ed Clancy, Director of Public Relations, 417 S. Hill St., Los Angeles 13.

CALIFORNIA HEART ASSOCIATION ANNUAL MEETING AND SCIENTIFIC SESSION, La Playa Hotel, Carmel, May 18 to 20, 1956.
Contact: Alan Croft Blanchard, field director, California Heart Association, 1428 Bush Street, San Francisco 9.

WESTERN BRANCH, AMERICAN PUBLIC HEALTH ASSOCIATION Annual Meeting, Salt Lake City, Utah, May 30 to June 2.
Contact: Mrs. L. Amy Darter, secretary-treasurer, at State Public Health, 2151 Berkeley Way, Berkeley 4, California.

**For registration or information, contact:* Gertrude Jones, M.D., Children's Hospital, San Francisco.

†*For information, contact:* John B. Flick, M.D., 255 S. Fifteenth Street, Philadelphia 2, Pa.

INFORMATION

Maternity Practices in Hospitals in California

THE FOLLOWING REPORT summarizes two regional conferences on maternity and newborn care in general hospitals held in Santa Barbara, October 22 and 23, 1954, and in Sonoma, November 5 and 6, 1954. The purpose of these conferences was to analyze the laws, practices and traditions which establish prevailing standards of maternity care in general hospitals. The conferences resulted from recognition that present high standards of maternity care are accompanied by practices which tend to make inflexible the utilization of space and personnel in obstetrical services of general hospitals. This inflexibility tends to reduce the maximum utilization of physical facilities and staff, with resultant high patient-day costs.

Approximately 35 individuals participated in each conference. Participants included obstetricians, pediatricians, physicians in general practice, and in public health, hospital administrators, nurses in obstetrics, in administration and in public health, members of faculties of universities, and staff members of the State Department of Public Health.

In this report it should be recognized that there is unanimity that high standards of obstetrical care are essential, but unanimity does not always exist as to what is necessary to maintain these high standards. This report provides a compilation of the conferees' best judgment on major maternity practices in hospitals for the advice and guidance of those responsible for patient care in hospitals.

In some instances, conclusions reached by the conference are at variance with current legal requirements which are contained in regulations set forth by this Department under the Hospital Licensing Act. In other instances, while conclusions are not at variance with the law, physicians and hospital administrators may wish concurrence of national organizations, such as the Joint Commission on Accreditation of Hospitals and the American Academy of Obstetrics and the American Academy of Pediatrics, before modifying present practices to comply with conclusions reached in the conferences. The State Department of Public Health intends to bring conference recommendations which are contrary to current legal requirements before the Hospital Advisory Board and State Board of Public Health in public hearings to determine whether legal regulations should be changed to comply with conference recommendations. The Department also intends to bring conference recommendations to the official attention of technical groups such as the Joint Commission on Accreditation of Hospitals, American Academy of Pediatrics, and the American Academy of Obstetrics.

Copies of the complete report are available by writing the State Department of Public Health, 2151 Berkeley Way, Berkeley 4.

Conclusions and recommendations of the Conferences on Maternity Practices of Hospitals in California. Prepared by Bureau of Hospitals, State Department of Public Health.

BACKGROUND, OBJECTIVES, SCOPE, AND ORGANIZATION OF CONFERENCES

A. Background

Serious economic problems are created in operating obstetrical departments under currently accepted laws and customs because of low space utilization and limited flexibility of personnel in obstetrical departments of general hospitals. The State Department of Public Health became interested in certain traditions of maternity service—particularly the segregation of maternity patients from others in the hospital—in its activity of assisting communities throughout the state to plan and develop hospital facilities and through administration of the hospital licensing program.

Review of bed usage experience in existing hospitals disclosed low average occupancy in maternity departments, compared with a much higher occupancy in other hospital departments. Statistics in a recent study showed that average occupancy of acute hospitals in California is 74 per cent. In these hospitals, however, the average occupancy in the maternity service is 53 per cent and the average in other services is 78 per cent. If maternity departments could operate at 78 per cent capacity, 3,713 obstetrical beds properly distributed throughout the state could accommodate all maternity cases. At present there are 5,866 maternity beds, or approximately 2,000 more than could be utilized at an average occupancy rate of 78 per cent.

Based on State Department of Public Health estimates, California has only two-thirds of the general hospital facilities it needs to serve the state's rapidly increasing population. As new hospitals are built, they invariably include maternity departments. The economic consequences in cost of construction and operation of maternity facilities, which have relatively low utilization, are a serious matter which, over a number of future years, involves many millions of California residents' dollars.

Some maternity departments, because of their isolated location from other parts of the hospital, have actually closed. Because hospitals have been required to maintain separate delivery rooms and operating rooms, the low volume of deliveries in some hospitals has caused elaborately equipped delivery rooms to stand idle much of the time. Many hospitals reported that their maternity departments were consistently operating at a deficit.

Study shows that the same general problems exist in hospitals of all sizes, and while shortened patient stay has contributed to the problem of low maternity census, it is less significant in terms of long-range use of obstetrical area than the inflexibility which results from current practices. While some hospitals, in an effort to overcome the deficit and to meet the demand for more medical and surgical beds, have admitted other types of patients to the maternity section when the census in the department is low, others question the advisability of this practice. After consideration, it was determined working conferences would provide the best method of reviewing the problem and its many implications. It was thought that if experienced and well qualified participants were selected, certain conclusions regarding safe, efficient, and more economical care might be reached, and would point out the need for studies and further evaluation of certain practices and procedures.

B. Objectives of the Conference

Objectives for the conference were:

1. To develop suggestions and recommendations for high quality of maternity care;
2. To determine the importance of strict segregation of maternity departments;
3. To evaluate present procedures and practices from standpoint of safety, efficiency and economy;
4. To designate certain procedures or practices where further study is indicated;
5. To consider how recommendations of the conference can be implemented.

C. Scope and Organization of the Conference

In developing plans for these working conferences, the Department consulted a steering committee made up of representatives of the California Medical Association, Northern California Pediatric Society, California Hospital Association, California League for Nursing, California Conference of Local Health Officers, and University of California School of Public Health.

The number of participants selected for each conference by the organizations which they represented, included: Twelve physicians representing the California Medical Association, Academy of General Practice, California chapters, Academy of Pediatrics, Obstetrical Societies, and California Medical Schools. Five representatives of the California Hospital Association, five of the California League for Nursing, two of the California Osteopathic Association, two of the University of California School of Public Health, five of the California Conference of Local Health Officers and five of the State Department of Public Health.

The two-day conferences were organized so that the entire group met together at the opening session and the closing session, but the participants were divided into five small groups for the other meetings.

In the closing session, each group summarized and presented the conclusions of one of five discussion topics as they had been prepared by each of the other groups. By this method, it was possible to present all recommendations of all groups in a brief and concise manner in the limited time available.

RECOMMENDATIONS AND CONCLUSIONS OF THE CONFERENCE

The following report summarizes the conclusions and recommendations of the conference and is divided into the five areas of maternity service, in accordance with the organization of the conference:

I. Admission and Labor
II. Delivery
III. Maternity Patient Unit
IV. Nursery
V. Rooming-in

A. Admission and Labor

Admission Routines. Most of the groups at both conferences believed that no special dangers were encountered in the usual admission procedures of maternity patients, regardless of the stage of labor. Several groups stressed the importance of avoiding delay of admission, particularly in county hospitals where maternity patients are admitted through the same channels as all other patients. While the danger of contracting an infection in the process of admission is probably minimal, there is psychological reason, as well as obstetrical reasons, for getting the patient admitted to her room or to the labor room expeditiously.

Dealing with Patients with Communicable Disease. How to deal with a patient who, upon admission, has a communicable disease or has been exposed to one, was not agreed on by all participants. While all agreed that isolation of some kind was necessary, there was divided opinion as to whether she should be isolated in or out of the maternity unit.

Whether the patient actually had the communicable disease or had merely been exposed to com-

municable disease should be considered. If the exposure was to a disease not ordinarily contracted by adults, the need for isolation would be lessened. The degree of transmissibility, the available facilities within the hospital (both in and out of the maternity department) and the availability of skilled nursing personnel were factors that were mentioned at the conference. Conference opinion was that placing the patient in a private room under observation in the maternity unit after delivery would be adequate in most cases. Many members felt that delivery could be carried out in the delivery room without creating hazards for other patients.

It was recommended in both conferences that a woman exposed to communicable disease who delivered prior to admission constituted minimal danger and should be admitted to the maternity department. Observation for a period of 24 to 48 hours in a single room in the maternity unit would be adequate to protect the other patients from possible infection. Rooming-in was suggested as one way of handling the infant during this observation period. The suspect nursery within the unit was the other possibility.

Labor Rooms. It was agreed by the participants that labor rooms are desirable for all hospitals accepting maternity patients, although not from the standpoint of protection from infection. Technical efficiency, convenience for doctors and nurses, and psychological benefits to the patients were listed as reasons for labor rooms.

Ideally, the labor rooms should be in close proximity to the delivery room, but the conferees recognized the advantages of having labor rooms close to the patient area in small hospitals where there is no regularly assigned labor room personnel, even though physical segregation of the labor patient from other patients also is desirable.

Staffing the Labor Rooms. It was recommended that the best available qualified nursing personnel be used in the care of patients in labor. All conferees expressed the importance of this phase of maternity care and stated that when skilled personnel is limited, it should be utilized in the labor room.

Presence of Husband in Labor Room. Both conferences recommended that the husband should not be excluded from the labor room if his presence is acceptable to the physician and the patient. Unless the labor patient is in a single room, some means of maintaining patient privacy is desirable.

Whether it is necessary for the visitor to be gowned was not resolved. Some did not feel that gowning is necessary from the standpoint of preventing infection, but did feel that it would serve a psychological purpose.

B. Delivery

Separate Delivery Rooms. It was recommended that separate delivery rooms be required in all but the very small hospitals. Reasons for this conference recommendation were not based on preventing the spread of infection, but rather for technical efficiency, convenience, availability, ease of staffing, and maintaining harmonious staff relationships. Most conferees agreed that since sterile aseptic technique is observed in surgical procedures and in deliveries, there is no serious danger of spreading infection to either the maternity patient or surgical patient if the same room is used. It was felt that in hospitals where space could always be available for deliveries, some flexibility should be provided so that delivery rooms and operating rooms might be used for whatever procedure was necessary.

Separate Clean-Up Rooms. It was recommended by part of the groups that separate clean-up rooms for operating rooms and delivery rooms should be provided. Where separate staffs are maintained, it would be particularly desirable to maintain separate work rooms. One reason given was the possibility of spreading infection from surgical to obstetrical patients.

Separate Lockers, Toilets and Showers. It was recommended that separate lockers, toilets and showers were not necessary for surgical and obstetrical personnel, either doctors or nurses.

Nursing Staff in Delivery Room. It was generally agreed that while a specialized nursing staff does assure safer and more expert care, no infection hazards are created when the same staff serves delivery and surgery or the delivery room and patient units. Good technique of personnel was stressed as being of most importance.

Presence of Husband in Delivery Room. It was generally agreed in the conferences that the husband or any other relative of the patient should not be allowed in the delivery room. However, there were some conferees who held the opposite view.

Delivery Room Techniques. It was recommended that the present techniques and procedures usually followed in delivery rooms were adequate and desirable. None were listed as being too rigid or too lax.

C. Maternity Patient Unit

Segregation From Other Patients. It was generally recommended by the conferences that absolute segregation from other patients is not necessary for safe and adequate maternity care. Most members agreed that segregation is desirable for efficiency of the service. All members stressed the importance of good technique by personnel, regardless of whether or not the patient area is segregated.

A flexible overlap of obstetrical beds with other beds is possible, but the careful selection of other types of patients was repeatedly stressed. This careful selection would limit the types of patients which can be admitted to the same nursing unit.

One group stressed that maternity patients be given priority in the beds designated as "flexible."

Separate Patient Services. It was recommended that in a flexible unit where other types of patients are admitted, the part of that unit which is used for nonmaternity patients need not be provided with separate utility rooms, nurses' station, showers and toilets; however, all patients should be provided with individual equipment such as bedpans, thermometers, washbasins and emesis basins.

Separate Nursing Personnel. All conferees were of the opinion that separate nursing personnel for obstetrical and nonobstetrical patients was highly desirable when both types of patients were in the same unit. The majority of participants felt, however, that where the patients have been carefully selected and there is good technique, safe care can be provided if the same personnel cares for all the patients in the unit.

Traffic Through Maternity Department. It was generally agreed that traffic (visitor and service) through a maternity department does not constitute an infection hazard to maternity patients to any greater degree than to other kinds of patients.

Patients with Postpartum Infections. It was recommended that a patient who had postpartum infection should be retained in the maternity department and should be isolated in a single or semiprivate room. An exception to this practice would be made in the case of the highly transmissible diseases such as chickenpox and measles.

Visitors. It was recommended that visitors should be limited, as much as possible, to include as a minimum the father, and as a maximum not more than two visitors on any one visit. Hours should be restricted and no visitors under 16 years of age should be permitted. Gowning and masking of visitors were not considered necessary.

Visitors should be educated to the objectives of visiting regulations. Use of posters, classes, pamphlets, displays, etc., was suggested as a possible way to accomplish these objectives.

Evaluation of Postpartum Routines. It was recommended that postpartum routines be reviewed and further evaluated. Handwashing technique was stressed by both conferences as being of greatest importance in preventing spread of infection.

D. Nursery

Capacity of Nursery. It was recommended that nursery units should be designed to accommodate eight or nine bassinets. The reasons for the small units were to limit the number of contacts in the event that infection should occur in a nursery and to limit the number of infants who will be cared for by one member of the nursing staff.

Location of Nursery in Maternity Unit. It was recommended that the nursery should be in close proximity to the postpartum rooms. In order that nurseries not be disturbing to postpartum patients, the importance of soundproofing was expressed.

Nurseries for Observation. It was recommended that observation nurseries for babies suspected of having disease should be provided within the maternity unit, regardless of the size of the hospital.

Infants with Diagnosed Infections. It was recommended that infants with diagnosed infection should be immediately transferred from the maternity unit and isolated.

Physical Examinations of Nursery Personnel. It was recommended that all personnel assigned to care of newborn should be properly screened and should have periodic and annual physical examinations, including chest x-rays. General agreement as to need for inclusion of stool cultures and throat cultures in the examination was not reached.

Importance of Good Technique. The consensus was that with good technique the same personnel could care for infants in the "suspect" nursery and in the normal nursery. It was further agreed that the same nursing personnel could care for mothers and the newborn infants.

Return of Infants to Normal Nursery. It was recommended that newborn infants taken from the normal nursery to x-ray rooms or surgery can be safely returned to the normal nursery without endangering the other infants. Some of the participants urged that this practice be followed if adequate pediatric facilities are not available in the hospital. That the "suspect" nursery might be used to accommodate infants being returned from x-ray or surgery was also suggested.

Handwashing Procedure. It was recommended that the procedure of handwashing before and after handling each infant should receive the most emphasis for safe infant care. The conferees recognized that many inconsistencies in this procedure exist, such as duration of handwashing, method of rinsing, drying, and type of soap or detergent used, and it was recommended that this procedure be standardized.

Gowns and Caps. It was recommended that all personnel who enter the nursery should be required to wear gowns and have some type of hair restraint (net or caps).

Masks. It was recommended by most conferees that personnel working regularly in nurseries should not be required to wear masks. Some members,

however, considered masks necessary for all nursery personnel. Most were agreed that personnel who entered the nurseries only occasionally, such as maids, orderlies and doctors, should be required to wear masks.

Preparation of Infant Formulas. It was recommended that any clean area convenient to the maternity section and having the necessary facilities may be used for preparation of infant formulas. Furthermore, it was agreed that formulas for all departments, including maternity, communicable disease and pediatrics can be prepared in the same area, even if that area is in the maternity unit. Sterilizing the contaminated bottles from pediatric or other services before they are returned to the obstetrical formula room was considered necessary.

Terminal Heat Method. It was recommended by most of the participants that formulas be prepared by the terminal heat method. However, some members believed the aseptic method was acceptable, particularly since some formula substances could not be subjected to the terminal heat method. Trained personnel under professional supervision is necessary for formula preparation, regardless of the method.

E. Rooming-In

This area of maternity practice was included in order that its influence be presented to the working conference. Because rooming-in in itself is not acceptable by some groups, and is a controversial subject, it was not intended that approval or disapproval of the practice should be sought by the conference. The recommendations, however, reflect the group members' attitude toward the practice, and not much agreement was reached.

Lack of experience with the practice by many members of the group can also be held responsible for the lack of conclusions. The following generalizations are offered as an outcome of the discussions:

It was generally recommended that rooming-in units should consist of quarters for about four mothers and four babies. This was based on the idea that one nurse could probably care for and supervise the care of that number. One group of conferees expressed the idea that single rooms are more adaptable to rooming-in than multi-patient rooms. It was recommended that handwashing facilities be available in every rooming-in unit. It was recommended that nursing personnel be especially trained and interested in the aims and objectives of rooming-in. It was recommended that where rooming-in is practiced, there should be but one visitor—the father or some designated alternate. There was no agreement as to whether or not rooming-in infants can be returned to the normal nursery when it is necessary to remove the baby from its mother. All agreed that it might be a desirable practice to segregate rooming-in infants from infants in the conventional nursery to assure safe care, but not all conferees felt the practice was necessary.

FURTHER STUDY

The following subjects were listed by the conferees as warranting further study:

1. Admitting routines.
2. Rectal examinations of labor patients.
3. Masking by personnel in labor room.
4. Medical and nursing routines in labor room.
5. Single versus multiple patient labor rooms.
6. Ratio of number of labor beds to delivery rooms to maternity beds.
7. Psychological aspects of labor.
8. Anesthesia practices in delivery room.
9. Identification practices of newborn.
10. Cord techniques.
11. Size of nurseries.
12. Types of containers for formulas.
13. Effectiveness of "hand dips" in nurseries.
14. Methods of formula preparation.
15. Breast care routines.
16. Perineal care routines.
17. Early ambulation of postpartum patient.
18. Rooming-in.

THE PHYSICIAN'S *Bookshelf*

CARDIAC AUSCULTATION INCLUDING AUDIO-VISUAL PRINCIPLES—By J. Scott Butterworth, M.D., Associate Professor of Medicine, New York University Post-Graduate Medical School, New York, Maurice R. Chassin, M.D., Assistant Professor of Clinical Medicine, New York University Post-Graduate Medical School, and Robert McGrath, M.D., Associate Professor of Clinical Medicine, New York University Post-Graduate Medical School. Grune and Stratton, Inc., New York, 1955. 111 pages, $4.50.

This small volume was apparently written with two objectives in view: (1) To present the author's technic of the audiovisual method of teaching phonocardiography. (2) To summarize some practical, clinical aspects of cardiac auscultation.

The authors believe that audiovisual methods are needed to teach auscultation because of "the almost intolerable boredom of the student and instructor while waiting for others to listen" and "the reluctance of students to time sounds and murmurs." One may wonder if these are valid reasons for the employment of audiovisual aids. Whether these technics actually improve the students' ability in clinical auscultations at the bedside or merely provide a novelty in the classroom is not commented upon. The audiovisual instrument employed contains a large oscilloscopic screen upon which phonocardiograms can be viewed by a group of students while listening to the sounds through electronic stethoscopes. Tape recordings can be made at the same time to be played back and studied later. The method of using the instrument and the elementary physics of heart sounds are well described.

The authors have failed to attain their second objective for the reader is presented with no more information regarding clinical auscultation than is available in most current textbooks of physical diagnosis. Certain topics of distinct clinical value such as gallop rhythm are superficially treated and there is a notable lack of attention paid to recent studies of the heart sounds in mitral stenosis, pulmonary hypertension and congenital heart disease. The illustrations of phonocardiograms are poor because of the lack of any simultaneous reference tracings such as the electrocardiogram, pulse or apex impulse. Timing lines are absent.

This volume should be of interest to those who wish to employ audiovisual aids in the teaching of clinical auscultation and physicians who are interested in heart sounds.

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THE CARE OF YOUR SKIN—Herbert Lawrence, M.D. Little, Brown and Company, Boston, 1955. $2.50.

While not indicated by the title, this book concerns itself with the problems of acne. It is easily readable and written in a manner which should be understandable to the adolescent and young adult.

The author handles adequately, the emotional and psychologic impact of this disease on the personality of an individual at a stage in life in which facial appearance is most important. The author also suggests simple measures to properly meet this personality disturbance. At the same time, etiologic factors, diet, popular misconceptions and simple treatment are not neglected.

The book definitely has a place in the physician's therapeutic regimen, especially for patients who desire detailed information regarding the cause, prognosis and treatment of this disease.

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THE HYPOPHYSEAL GROWTH HORMONE, NATURE AND ACTIONS—Editors Richmond W. Smith, Jr., M.D., Physician-in-Charge, Division of Endrocrinology, Henry Ford Hospital, Detroit. Oliver H. Gaebler, M.D., Head, Biochemistry Department, Edsel B. Ford Institute for Medical Research, Detroit; and C.N.H. Long, M.D., Sterling Professor of Physiology, Yale University School of Medicine, New Haven. The Blakiston Division, McGraw-Hill Book Company, Inc., New York, 1955. 576 pages, $12.00.

At the Henry Ford Hospital in Detroit, Michigan, on October 27, 28, and 29, 1954, a group of 300 investigators from medical research laboratories in many countries, gathered together to discuss the present status of Hypophyseal Growth Hormone. The results of these three days of discussion are presented in this volume, which contains the text of 30 papers, complete with charts and figures, as well as the general discussion of the reports. The presentations were divided arbitrarily into five sections:

1. The Bioassay Preparation and Properties of Growth Hormone.

2. The effects of Growth Hormone on certain tissues such as bone, joints and kidneys.

3. The effect of Growth Hormone on the utilization of protein, carbohydrate and fat as sources of energy.

4. The effects of Growth Hormone on certain enzyme systems.

5. The effect of Growth Hormone on lactation in cows, and on growth in humans.

The symposium participants, all leaders in Growth Hormone research, presented data and concepts obtained from recent original investigations. Additional data and ideas were added in the general discussions. Thus, the most recent knowledge of the nature and actions of Hypophyseal Growth Hormone is made generally available. The reports cover a variety of Growth Hormone effects, but the studies of the relationship of Growth Hormone to insulin activity and to Diabetes Mellitus, as well as the effects of Growth Hormone on normal, dwarfed and postsurgical human subjects will be of great general interest.

These reports will be of most interest to the clinical investigator, and to those interested in tissue growth. We do not know yet the full importance of Growth Hormone in clinical medicine, but this symposium will represent a landmark and a reference point on the road to a greater understanding of this fascinating and elusive hormone.